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THE TREATMENT OF PORTAL HYPERTENSION

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Although recent surgical advances have resulted in a considerable measure of success in the management of portal hypertension, this condition remains a serious challenge because of its complexity, grave complications, and high mortality rate. Many of the technical difficulties which may be encountered can be dealt with successfully as a result of the studies of Whipple,[10] Blakemore,[2] Rousselot,[8] Linton,[4] Berman,[1] Rienhoff,[6] and others. However, the clinical problems presented by patients with this lesion are not confined to the hypertension within part or all of the portal bed nor the complications of severe hemorrhage from esophageal varices and ascites. For a variety of reasons, patients with this syndrome are poor-risk patients in the older age groups. Severe disease of the liver associated with markedly impaired liver function may persist and may progress postoperatively to the point of invalidism or death even though the hypertension has been corrected. In addition, hepatic coma may occur postoperatively; varices may persist; hemorrhagic tendencies from hypoprothrombinemia or thrombocytopenia may develop, or failure of the portacaval shunt may develop from delayed or secondary thrombosis.

In many instances confusion persists as to which surgical procedure is indicated for the treatment of a given patient. The more recent operative procedures which have been devised for the correction of portal hypertension include thoracotomy for intraluminal or extraluminal ligation of bleeding esophageal varices, portacaval shunt, splenorenal shunt, mesenterocaval shunt, the Phemis-

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Presented during the Birmingham Assembly of The Southeastern Surgical Congress, March 8–11, 1954, Birmingham, Alabama.

TABLE I

Portal hypertension, original surgical procedures used

Type of Operation	No. of Cases
Splenectomy	4
Splenorenal anastomosis	7
Portacaval anastomosis	4
Mesenterocaval anastomosis	1
Hepatic and splenic artery ligation	9
Hepatic, splenic and left gastric artery ligation	9
Total	34

ter[5] procedure with resection of the lower esophagus and upper stomach, and ligation of the hepatic, splenic, and left gastric arteries.

Our experience in 34 cases of portal hypertension, and a comparison of the results obtained with shunt procedures and arterial ligations are the basis of this report. The nature, causes, and diagnosis of this condition have been excellently described in previous reports.[2, 4, 8]

MATERIAL AND METHODS

During the past seven years a total of 34 patients with portal hypertension have been treated surgically by members of the Surgical Department of the University of Cincinnati (table I). Four patients were treated by splenectomy alone. Twelve were subjected primarily to venous shunt procedures, 7 of which were splenorenal, 4 portacaval, and 1 superior mesenteroportal. The remaining 18 had ligations of the hepatic and splenic arteries with or without ligations of the left gastric artery during the past three years. These cases, and the results of the therapy to date, are being studied in an effort to derive any information which would aim in assessing the value and limitations of the different procedures.

The causes of the portal hypertension in these cases were portal cirrhosis in 20 patients, post necrotic cirrhosis in 7, hepar lobatum in 1, extrahepatic venous

TABLE II

Location and cause of portal block

Location of Portal Block	No. of Cases	Cause of Portal Block
Splenectomy cases		
Extrahepatic	3	Thrombosis of portal or splenic vein
Intra and extrahepatic	1	Cirrhosis and thrombosis of portal vein
Venous shunt cases		
Intrahepatic	5	Portal cirrhosis
	4	Postnecrotic cirrhosis
	1	Hepar lobatum
Extrahepatic	2	Thrombosis portal vein
Arterial ligation cases		
Intrahepatic	15	Portal cirrhosis
	3	Postnecrotic cirrhosis

TABLE III

Incidence of preoperative hemorrhage from esophageal varices and of ascites

Operation	No. of Cases	Preoperative Incidence (Cases)		
		Hemorrhage only	Ascites only	Hemorrhage & ascites
Splenectomy	4	3	0	1
Venous shunt	12	9	0	3
Arterial ligation	18	4	1	13

thrombosis in 5, and cirrhosis and extrahepatic venous thrombosis in 1 patient (table II). The case of portal hypertension produced by syphilitic hepar lobatum is apparently unique in that we have been unable to find a similar reported case.

The ages of the patients varied from 11 to 69, the average being 36.7 years for the splenectomy patients, 40.5 years for those treated by shunt, and 46.7 for those treated by arterial ligation.

Of the 12 patients treated by one of the shunt procedures and of the 4 treated

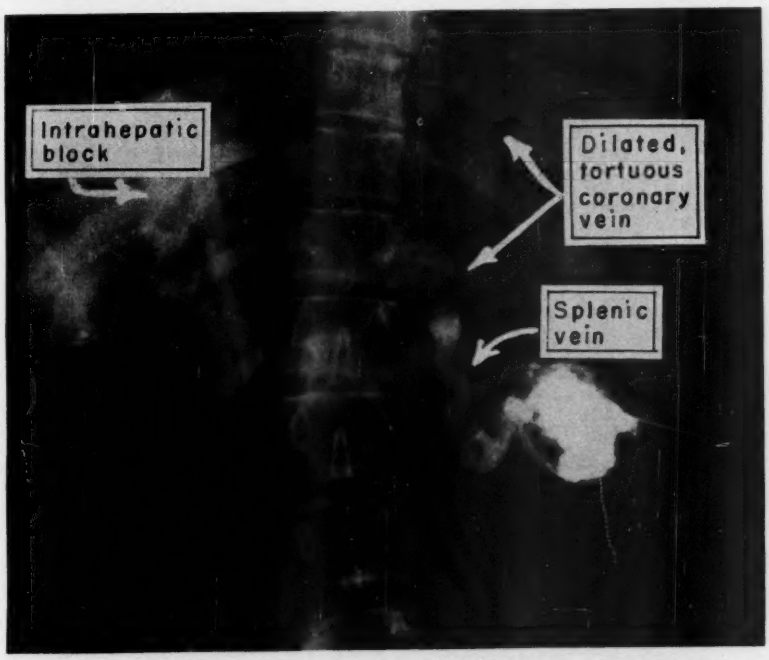

FIG. 1. Portal venogram taken March 19, 1953 after percutaneous injection of 20 cc. of Neo-Iopax (70 per cent solution) through a 19 gauge stylet needle immediately before operation in a 23 year old white woman with portal hypertension and recurrent hemorrhage from esophageal varices. Note that portal block is intrahepatic, that the coronary veins are tortuous and dilated, and that both the portal and splenic veins are large and patent. A splenorenal anastomosis was done successfully thereafter.

by splenectomy, all had had two or more episodes of bleeding preoperatively. Of those undergoing hepatic artery ligation, 1 had ascites only, 4 had hemorrhage only, and 13 had varying degrees of ascites and severe hemorrhage from esophageal varices (table III). In general those patients subjected to arterial ligation were more seriously ill and much greater surgical risks.

In an effort to determine preoperatively the site of the portal block, each patient was given a complete physical examination and routine laboratory work, as well as the following tests: blood urea nitrogen, prothrombin time, thymol turbidity, serum bilirubin, serum protein, bromsulfalein, cephalin flocculation, and roentgenographic examination after barium swallow. All but 5 of the patients also had liver biopsies.

In the last 3 patients having venous shunts, visualization of the portal vein immediately preoperatively was done by intrasplenic infusion of 20 cc. of Neo-Iopax (70 per cent solution) through a 19 gauge stylet needle according to the method of Leger[3] and Rousselot,[9] (fig. 1). This proved to be quite helpful in determining the point of portal block, the procedure indicated, and the site of the incision (fig. 2).

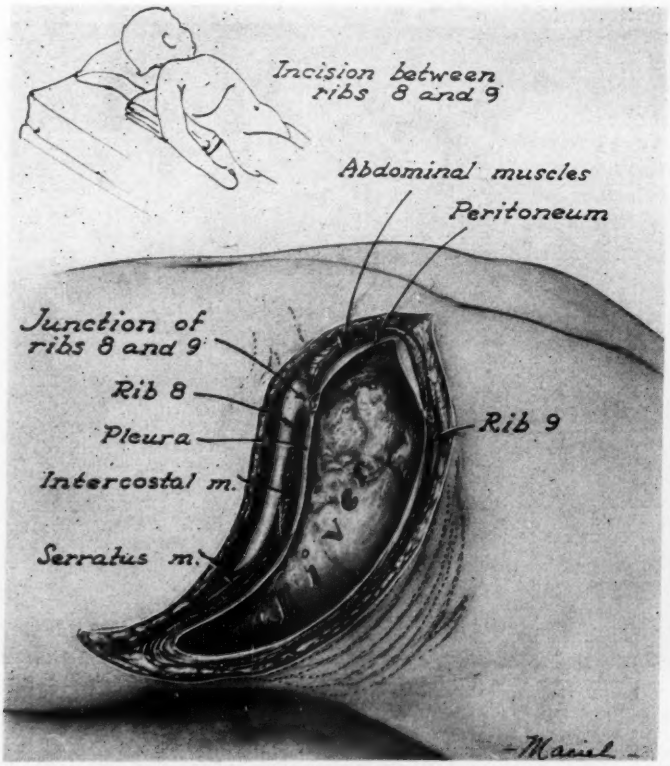

FIG. 2. Combined thoracoabdominal incision used on the right side for portacaval shunt. It has been used for splenorenal shunt on the left side in our patients also.

If the block was intrahepatic and the portal and splenic veins were intact, either a portacaval or splenorenal shunt could be decided upon (fig. 3a, b). A splenorenal shunt was decided upon in presence of an obliterated portal vein but an intact splenic vein.

Postoperatively the patients have been observed and the results of the operative procedures assessed.

RESULTS

It is very difficult to evaluate the results of treatment in patients with portal hypertension. However, in comparing the results obtained in the patients treated by the various methods, several interesting observations were made. The immediate postoperative mortality rate within three weeks after operation was zero in those patients undergoing splenectomy or venous shunt procedures. On the other hand, the immediate postoperative mortality rate in the patients treated with hepatic and splenic arterial ligations was 11.2 per cent. In addition five more deaths occurred among the arterial ligation patients within four and one-half months after operation (table IV). One of these deaths was not related to the portal hypertension, but the remaining four were. An eighth death occurred in a patient who had developed recurrent bleeding 25 months after arterial ligation and who then had received a portacaval shunt 19 months after ligation.

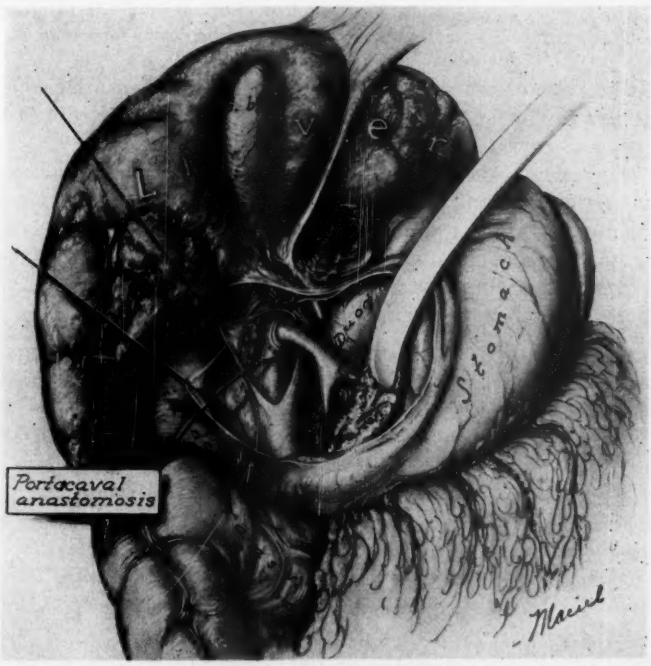

FIG. 3a. Drawing showing completed portacaval anastomosis and the relationship of portal vein and vena cava to the adjacent viscera as seen through a right thoracoabdominal incision.

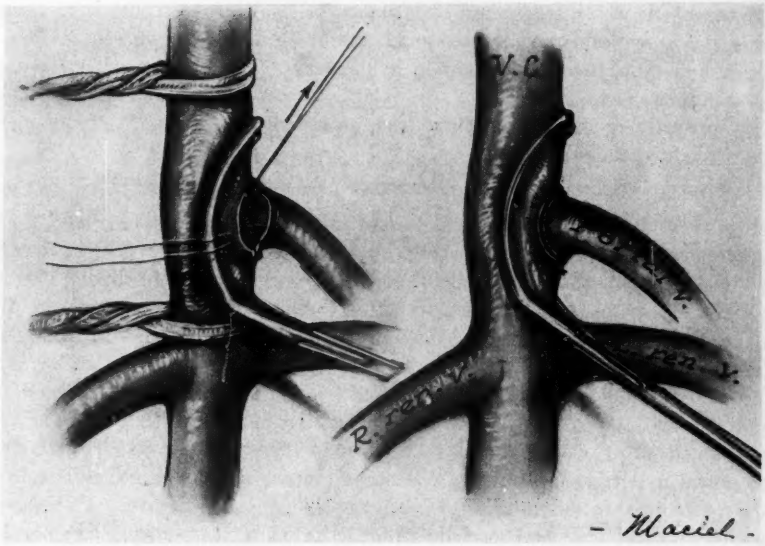

FIG. 3b. Drawing illustrating the technic of portacaval anastomosis used in our more recent cases of portal hypertension. Suture material was no. 00000 arterial silk. The clamp is a curved Pott's clamp. Essentially the same technic has been used for splenorenal shunts.

The latter was also unsuccessful, and the patient died. The number of deaths in the patients having ligations gives an over-all mortality rate of 44.4 per cent for a three year period after operation.

All of the patients who received a splenorenal shunt still are alive. Two of the other 4 patients having portacaval shunt died, 1 as the result of a strangulated diaphragmatic hernia and the other of recurrent gastrointestinal hemorrhage (fig. 4). This number of deaths in the 12 patients treated by venous shunt gives an over-all mortality rate of 16.6 per cent.

The causes of death in the arterial ligation cases were as follows:

1. Severe shock	Death within 8 hours postoperatively
2. Hepatic coma	Death within 4 days postoperatively
3. Recurrent esophageal bleeding	Death within 4 weeks postoperatively
4. Multiple lung abscesses	Death within 6 weeks postoperatively
5. Splenic infarction with subphrenic abscess	Death 3 months postoperatively
6. Hepatic failure	Death in 4½ months postoperatively
7. Hepatic failure with generalized capillary bleeding	Death in 5 weeks postoperatively
8. Recurrent hemorrhage	Death in 25 months after ligation. Portacaval shunt had also been done 19 months after ligation.

The fourth death was not related to the operation nor the liver disease.

Recurrent hemorrhage from esophageal varices has occurred in 6 of the 18

TABLE IV

Results of surgical treatment

Follow-up, 11 to 80 months

Type of Treatment	No. of Cases	Mortality Rate Within			Recurrent Bleeding in Survivors	Recurrent Ascites in Survivors	Remarks
		3 wks.	4½ mos.	Later			
Splenectomy	4	0	0	0	3	0	Massive bleeding in 3
Splenorenal anastomosis	7	0	0	0	2	0	2 episodes of massive bleeding postoperative in 1 patient, and 1 episode of slight hematemesis in 1 patient.
Portacaval	4	0	0	2	1	0	Death from hemorrhage 11 months later—1 patient. Death from strangulated diaphragmatic hernia—1 patient 6 months later.
Mesentero-caval	1	0	0	0	1		Slight tarry stools for 2 days, 4 months after operation.
Hepatic and splenic a/o left gastric artery ligation	18	2	0	0	0		Severe shock in 1 patient, hepatic coma in 1 patient.
			5		1		Hepatic failure in 2 patients, recurrent bleeding in 1 patient, lung abscess in 1 patient, splenic abscess in 1 patient.
				1*	1		
					4	0	4 patients have had 2 or more episodes of esophageal hemorrhage in the 10 survivors.

* Recurrent hemorrhage in 26 months.

patients treated by arterial ligation (33.3 per cent). In 2 it caused death while in the remaining 4 it varied from minor to moderate in severity. In another patient, generalized bleeding developed from all of the mucous membranes during hepatic coma due to liver failure. Recurrent and massive bleeding has developed in three of the four patients having splenectomy alone. In 1 of these a secondary portacaval shunt has been done successfully and in the other 2 a Phemister procedure has been necessary when post-thrombotic obliteration of the portal vein was found (table V).

Of the 13 patients who originally had ascites and esophageal bleeding before arterial ligation, 11 survived a postoperative period of four and one-half months and none of these showed a recurrence of the ascites, and 6 have not had further bleeding from the esophageal varices. Of the 3 patients with ascites and hemorrhage before their venous shunt, 1 developed a recurrent ascites which was finally controlled by medical management.

FIG. 4. Autopsy specimen of a 45 year old white woman, showing intact and patent portacaval shunt which had been done successfully eight months before for portal hypertension with recurrent hemorrhage. Death was unrelated to portal hypertension and was caused by a strangulated diaphragmatic hernia.

The mortality rate was higher in the patients with postnecrotic cirrhosis who had hepatic arterial ligation. All 3 patients with this lesion died within three months of severe liver failure. In contrast, 3 of the 4 patients with postnecrotic cirrhosis treated by venous shunt have survived after the procedure.

The data on liver function tests of these patients is voluminous but incomplete at this time. It will be the basis of another and later report.

The portal pressure, as measured at the operating table, varied between 235 and 460 mm. of water in the splenectomy and venous shunt groups, and 240 and 440 mm. of water in the group treated by arterial ligation. Pressures less than 300 mm. were found in 4 patients who had two or more episodes of bleeding from esophageal varices, the levels being 235, 240, 265, and 280 mm.

In addition to the complications enumerated above in the fatal cases, others

TABLE V

Secondary surgical procedures

Original Operation	Secondary Operation	No. of Cases	Interval
Splenectomy	Phemister operation	2	23 months 8 years
Hepatic and splenic artery ligation	Portacaval anastomosis	1	23 months
Hepatic and splenic artery ligation	Left gastric artery ligation	1	25 months

encountered were coma, protracted hypoproteinemia, atelectasis, laryngeal edema, delirium tremens, subcutaneous emphysema, and postoperative wound bleeding after heparinization.

The immediate postoperative course was uncomplicated in 21 of the 34 patients.

COMMENTS

A study of these results has yielded certain impressions and has indicated trends. The poor results obtained with splenectomy alone in the 4 patients with extrahepatic portal blocks with recurrence of severe bleeding in all but 1 patient to date, emphasizes the belief that splenectomy alone rarely should be done in the treatment of portal hypertension. Instead, a splenorenal venous shunt should be done at the time of splenectomy, in our opinion. Rousselot, Blakemore and Linton previously have emphasized that a splenectomy should not be done in patients with portal hypertension unless one is prepared to proceed with a splenorenal shunt. If it is not done, obliteration of the splenic vein may occur postoperatively and prevent a subsequent or lateral splenorenal procedure. In those patients with postphlebitic obliteration of the portal vein, a portacaval shunt likewise is impossible and the surgeon is faced with the necessity of doing a more difficult and tedious superior mesenterocaval anastomosis or a Phemister procedure. It is significant to note that a Phemister operation became necessary in 2 of the 4 patients treated originally by splenectomy. These patients are alive and in a fair state of health three years and one year postoperatively. There was one minor episode of recurrent hemorrhage in the patient operated upon one year ago.

In general, the results which we have obtained with the splenorenal and portacaval shunts have been superior to those obtained with hepatic and splenic arterial ligation which were done for portal hypertension with recurrent hemorrhage. The mortality rate has been considerably less, both in the immediate postoperative period and during the period of follow-up.

Although it must be remembered that the patients selected for hepatic and splenic arterial ligation were generally much greater risks than those selected for venous shunt, an immediate postoperative mortality rate of 11.2 per cent and an over-all mortality rate of 44.4 per cent for the arterial ligation group is significantly higher than an immediate mortality rate of zero and an over-all mortality rate of 16.6 per cent in the venous shunt group.

A recurrence of hemorrhage from esophageal or rectal varices occurred in 6 of the 16 patients (37.5 per cent) surviving the immediate postoperative period of three weeks after arterial ligation, while the same complication has developed in 4 of 12 patients (33.3 per cent) after venous shunting. In 2 of the latter 4 patients, however, the bleeding has been minimal and limited to small hematemesis. This suggests that the results in patients with portal hypertension and hemorrhage have been better in the patients treated by splenorenal or portacaval shunts, although it must be remembered that 6 patients of the original 18 treated by arterial ligations have had no bleeding since operation 24 to 33 months ago.

Considering the higher mortality rate obtained with the arterial ligation procedure, however, the operation of choice appears to be a splenorenal or portacaval shunt, particularly in the greater-risk patients.

The value of hepatic arterial ligation in cases of ascites, however, appears possibly to be greater than that of a venous shunt. It is interesting to note that none of these patients developed recurrent ascites if they survived the postoperative period of four and one-half months. This possibility needs further study for clarification. Similar promising results have been reported by Rienhoff and Woods[7] in the treatment of ascites.

The high mortality rate obtained in the cases of postnecrotic cirrhosis treated by hepatic artery ligation suggests that this procedure is too hazardous for this condition. The development of severe and fatal liver insufficiency three to five weeks postoperatively with coma, shock, massive ascites, uremia, generalized bleeding from all mucous membrane surfaces and terminal pneumonia is a complication which will preclude the use of hepatic artery ligation in our cases of postnecrotic cirrhosis in the future.

The value of excellent medical assistance in the diagnosis and treatment of these patients cannot be overemphasized in our opinion.

SUMMARY

The surgical treatment of portal hypertension is limited to procedures designed to prevent further hemorrhage from esophageal varices in patients with intrahepatic and extrahepatic portal vein obstruction.

On the basis of the results which have been reported in 34 of our cases of portal hypertension, the operative formation of a venous shunt, either between the portal vein and the vena cava or between the splenic and renal veins, is the most satisfactory and safest procedure for the treatment of recurrent hemorrhage from esophageal varices. In our small number of cases, the splenorenal shunt has given the best results. Hepatic and splenic arterial ligation has given sporadic and irregular results in patients with portal hypertension and hemorrhage. In one-third of the patients, the results have been excellent. However, the higher over-all mortality rate obtained with the arterial ligations has made it a more dangerous procedure than a venous shunt.

Those patients with severe and advanced cirrhosis are much greater surgical risks than those with extrahepatic block. The presence of postnecrotic cirrhosis has been associated with a very high mortality rate in patients subjected to hepatic arterial ligation, and probably should be considered a contraindication to this operation.

In the management of patients with portal hypertension there is suggestive evidence that hepatic and splenic arterial ligation is a valuable method of treatment, although this is very difficult to evaluate.

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SURGICAL TREATMENT OF COARCTATION OF THE AORTA IN THE ADULT

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Since Crafoord's first successful case in 1944, several reports have appeared in the world's literature relative to the surgical management of coarctation of the aorta. The majority of these reports have been concerned with the treatment of this condition in childhood. In this report I would like to present our experience at the clinic with the surgical management of coarctation of the aorta in a small series of adults.

It is acknowledged that the presence of coarctation of the aorta is compatible with long life and with minimal symptoms. Students of this subject, however, repeatedly have stressed the early development of fatal complications. The average age of all patients at the time of death was 35 years in Maude Abbott's series. These patients usually die of the complications of hypertensive cardiovascular disease, although rupture of the aorta and subacute bacterial endocarditis may occur.

Twenty patients suffering from coarctation of the aorta, whose average age was 25 years, were encountered in 100 consecutive cases of congenital heart disease. The ages of the patients operated on in this series ranged from 21 to 36 years. Thirteen were males and 7 females. The majority of these patients had no symptoms whatever, and when symptoms were present, another cause usually could be found to account for them.

A cardiac murmur was present in every case in this series. This murmur frequently showed the typical transmission to the left paraspinal area just medial to the scapula. All of the patients had an elevation of the systolic blood pressure. In only 9 patients was an attempt made to secure a blood pressure reading in the legs. In these 9 patients the blood pressure was low or absent entirely. Two patients had a faint pulsation in the abdominal aorta, and 17 had none whatever. A few of the patients had palpable intercostal vessels, usually felt best near the scapula. In all but 1 patient in the group notching of the ribs was apparent in the roentgenogram. This sign is almost pathognomonic.

In some of the patients, angiocardiograms have been made using 70 per cent diodrast injected into a cubital vein. Although the dye may be considerably diluted, satisfactory visualization of the stricture can be obtained. We have not used direct arteriography. It is our belief that this diagnosis perhaps is the simplest of the common anomalies, and the chief reason for using angiocardiograms has been to determine the presence or absence of a descending aorta.

From the Department of Surgery, Lahey Clinic, Boston, Massachusetts.
Presented during the Birmingham Assembly of The Southeastern Surgical Congress, March 8–11, 1954, Birmingham, Alabama.

SURGICAL CONSIDERATIONS

The patients selected for surgery have had a serious stricture of the aorta. This has been inferred from the presence of severe hypertension in the arms and hypotension in the legs. Those with mild or moderate hypertension have not been operated upon. It is possible that we will regret this stand in future years.

It is probable that in the early teens, an average age of perhaps 12 years is ideal for surgical correction of coarctation. Operation should be deferred if possible until a sufficiently large and elastic aorta is available. If the operation is done at an earlier age the anastomotic suture lines might not develop with the developing aorta and a relative stricture might result with full growth. On the other hand, from adolescence on, atheromatous changes become increasingly evident in the average patient. Therefore, operation should not be delayed too long.

In general, these patients tend to be healthy, well developed, and even vigorous. They require very little preoperative preparation. Intratracheal anesthesia is used and two veins are cannulated in the operating room.

A wide exposure of the superior mediastinum is essential. The entire fifth rib is resected and the fourth and sixth ribs are divided posterior to their angles. A long and tedious dissection is required to open the chest. Huge, tortuous, thin-walled collateral vessels are present everywhere, particularly around the scapula, and a pint or even a liter of blood may be lost in the adult patient before the chest is opened widely.

The coarctation may be readily visualized by incising the mediastinal pleura over the descending aorta, over the arch, and over the left subclavian artery. The large size of the left subclavian artery is immediately noted although, as a rule, the descending aorta is relatively normal. One of the great problems in this operation is dealing with the intercostal vessels. It will be recalled that they are the chief avenues for collateral circulation. Through them in reverse flow the blood is restored to the distal aorta. These vessels are large, thin-walled, and pass almost immediately into the parietes. A very careful technic has been worked out to manage them. The distal end of the vessel is dissected to its point of disappearance into the intercostal muscles. Ordinarily, there will not be room to doubly tie and cut between, so a silk tie is placed as close to the aorta as possible and a clamp is placed on the distal artery. After the vessel has been divided, this end is transfixed and ligated. Very special care has to be taken to secure the right-sided intercostal arteries coming off the right posterior aspect of the aorta.

The aorta in the region of the coarctation and the angles between the great vessels are cleared of fascia and the vagus and recurrent laryngeal nerves are dissected out. Finally, the ligamentum arteriosum is divided. Occasionally this may actually be patent and has to be handled with care. Section of this structure will liberate the aorta and the coarctation may be excised. The point of resection should be placed so as to avoid any intercostal stumps that may interfere with the placement of sutures in the anastomosis. We have tried many

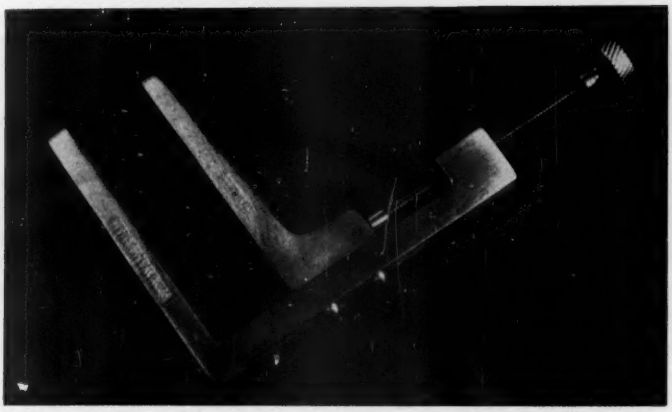

FIG. 1. Specially designed aortic clamp.

clamps and at the present time we use a specially designed clamp, on the Blalock principle, which has short handles to secure the proximal and distal aorta and the subclavian artery (Fig. 1).

In all but 3 patients end to end anastomosis was possible after resection. In our early experience with this work, the left subclavian artery was divided, brought down and anastomosed to the distal aorta in 3 patients.

A technic has been worked out for the performance of the anastomosis. We have believed with others that an eversion suture is safest. We have insisted, however, upon the use of interrupted sutures as a foundation for the anastomosis. We have used no. 000 Deknatel silk because we do not believe that no. 00000 Deknatel silk is strong enough for the adult aorta. The first suture is an everting mattress, including all layers, and tied on the proximal aortic side at the 2 o'clock position as viewed from the left side of the table. The first suture is the key. Manipulation of this will permit the critical and difficult medial corner to be sutured with precision. By passing this suture back and forth, the corner may be rounded and interrupted sutures are placed about a millimeter apart throughout the circumference of the aorta. The anterior suture line will evert itself if the beginning is correct. The sutures are so placed that the everted cuff of aorta will be sufficient to accommodate a very fine silk suture. This is a second layer and is continuous, broken in two or three places.

In none of our cases has collapse occurred when the clamps were removed. We have used a definite sequence and we remove the clamps very slowly. The following routine is suggested: The subclavian circulation should be kept shut off while the aortic clamps are removed. After the patient has accommodated to this first change in his circulation the clamp on the subclavian artery may be removed slowly. Thus it is possible to avoid opening two very large vascular channels simultaneously. We have at times noted a slight drop in blood pressure when the lung is re-expanded and the pulmonary vascular bed is opened.

RESULTS OF SURGERY

No serious complications were encountered in the postoperative period in any of these patients. Persistent atelectasis of the left upper lobe developed in 1 patient. These patients were kept at bed rest for a week; were allowed some activity for the second week, and were dismissed at the end of the third week.

One patient died two months after operation, having been readmitted to the hospital because of severe pain in the interscapular region. Roentgenologic examination revealed a soft tissue mass in the region of the anastomosis. This was interpreted as being an aneurysm. Dissection of this aneurysm with impend-ng rupture was diagnosed. This came in the form of hemorrhage into the left bronchus. Postmortem examination showed the formation of a false aneurysm with rupture secondary to separation of the suture line.

All of the remainder of this group of patients are asymptomatic and apparently well. They have been followed carefully and it is apparent that the blood pressure is greatly reduced. It usually is in the range of 140 to 150 mm. systolic and 80 to 90 mm. diastolic. No other cases of aneurysmal formation have been detected.

CONCLUSIONS

Twenty adult patients have been operated upon successfully for coarctation of the aorta. The results of these operations have been good to date.

One patient developed a false aneurysm due to rupture of the suture line. It will require several years experience to determine the true incidence of this complication. At present we believe that this complication should not deter us from advising operations upon patients with coarctation who have hypertension.

Later, with increasing experience, indications for surgical intervention may be extended or restricted as the results dictate.

PILONIDAL DISEASE

REVIEW OF LITERATURE AND A METHOD OF CLOSURE

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Numerous articles on pilonidal disease have resulted from the experiences gained by physicians in the Armed Services since the beginning of World War II. This situation is understandable when one considers the millions of young men who are called into the Service and the activities to which they are exposed. Pilonidal disease, despite its usual minor surgical appearance, manifests major surgical significance when treatment and permanent cure are undertaken. The magnitude of this condition is shown by Casberg (1949).[4] He reports 78,924 admissions to Army Hospitals from 1941 to 1945 inclusive for pilonidal disease. The navy lists 7,409 operations for this disease during the peace time years of 1947 to 1950. Our interest in this disease continues to be stimulated not only by its incidence but also by the variable results of treatment.

Probably the first description of the disease appeared in a letter written by Anderson (1847[1]) to the editor of the Boston Medical and Surgical Journal in which he described *"hair in an ulcer"* over the low sacral region. However, it was not until 1880 that the term *pilonidal* was employed by Hodges[11] when he appeared before the Boston Society for Medical Improvement.

Warren (1887) recognized the disease to be congenital. There have been two schools of thought relative to the embryonic formation of the cyst. Hermann and Tourneauz (1887), Mallory (1892) and Gage (1935[8]) all discuss the failure of closure of the medullary canal as being the cause, while Frere (1878) and Fox (1935[7]) speak of the invagination or embryonic infolding of the cutaneous epithelium. Currently, the latter theory seems to be the more popular. Mention also should be made of Stone (1931[19]) who noted the similarity between the cyst and preen glands in birds; and Kallet (1936[13]) who discussed the cyst as being an *embryonic rest of vestigial secondary sex gland.* Regardless of the exact etiologic factors, it is accepted that the condition is congenital and that the abnormality first becomes apparent in about 70 per cent of the patients between the ages of 16 to 35 years—the military age.

In conjunction with the discussion of etiology, it would be well to consider what is called a recurrent cyst or sinus; that is, the condition in which the cyst has been

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completely excised and is followed by another draining sinus. Emphasis is placed on complete excision of the cyst. The large number of recurrent sinuses observed during a period just prior to this study were due primarily to faulty closures and improper wound healing. In none of them did we find evidence of a true recurrent epithelial-lined cyst. This observation coincides with those of many others.[9] Thus the problem of recurrence seems to depend almost entirely upon defective closure of the sacral defect after complete excision of the cyst. The migration of hair into small skin separations at the distal angle of the wound near the anus is well recognized. Since most recurrent drainage tracts are found in this distal one third of the wound, it becomes apparent that proper handling of this portion of the wound alone would forestall numerous recurrences.

Numerous surgical procedures to correct this defect have been described during the past 15 years.[6, 16, 20] Basically all of the procedures used can be divided into two categories; the open and the closed method. The open method ultimately will result in healing following prolonged postoperative care. Pocketings in the deep granulation tissue must be guarded against as well as early bridging of skin edges. Either of these complications favors recurrences. The postoperative management of the open method on an out-patient status is not possible in the military environment and results in an extended period of hospitalization greatly exceeding the medical importance of the disease. The broad, hard, relatively avascular scars which result cause erosion and maceration of deep natal clefts and require constant treatment in the surgical out-patient clinic with occasional rehospitalization. The rigors of military life may possibly make this condition more common in the service than in civilian life.

The defects of the open method have kept us constantly on the lookout for a closed procedure which would properly consider the anatomic structure of this region and permit adequate excision, strict hemostasis, obliteration of dead space, closure without tension and construction of a vascular pad over the sacrum and coccyx.

If one closely studies this sacrococcygeal region of the body in action, it readily becomes apparent that it is a hub for physical activity. A study of the lines of stress in this area shows them to go in radial directions from the midline. An excellent article on this subject was reported by Harberson and Brintnall (1950).[10] Their skin incisions were patterned along these lines of stress.

Various skin incisions have been advocated for pilonidal cystectomy. Berman (1945)[2] tried a combined transverse and longitudinal incision forming four flaps which were approximated without tension. Brown (1945)[3] described an upper transverse incision combined with a lower 'V' incision. Lahey Clinic tried a skin pedicle flap; Cattel described a 'Y' shaped flap and Cohn (1943)[5] tried a 'U' shaped skin incision. A transverse elliptical incision has been used for small simple cysts. All of these modifications have been attempts to get away from the midline scar; lessen the stress on the wound, or save some skin and subcutaneous tissue.

In discussing the wound, consideration should be given to the four major factors in primary wound healing: asepsis, obliteration of dead space, good blood

supply and avoidance of tension. The dead space resulting from *en bloc* excision of the cyst, has the sacrococcygeal fascia for a floor, and the sloping gluteal muscles and fat for side walls. A review of the literature would indicate that the most commonly employed method of closure is, first, the undermining of the fat from the gluteal fascia, and second, placing wire sutures through skin, fat and sacrococcygeal fascia and tying these wires over pressure dressings. This procedure is an attempt to obliterate the dead space but the actual result is not immediately apparent. Another method, first described by Corp (1929), involves suture of the skin edges to the sacrococcygeal fascia. This type of closure deprives the sacrum and coccyx of a protective pad. Another procedure consists of dissecting out the cyst and saving all the fat and skin possible.

Shute, Smith, Levine and Burch (1943)[18] described a technic employing the gluteal muscle and fascia to cover the sacrum and coccyx and to fill the dead space between the nates. The same method was described by Miscall and Holder (1943)[15] a few months later. This method involved making longitudinal incisions into the gluteal fascia and muscle approximately 2.5 cm. from their attachment to the sacrum and coccyx. These medial musculo-fascial flaps were sutured across the midline. The lateral flaps were then undermined and sutured under tension in the midline over the medial flaps. The subcutaneous tissue and skin were then closed. Both groups of authors reported fairly good results with this technic. They undertook to cover the sacrococcygeal fascia and bone with a vascular pad of muscle. We believe that maneuver is most important because a direct fat to fascia closure of the defect with the resultant poor blood supply ends in unsatisfactory healing. Recurrences usually disclose an infected pocket lying directly on the sacrum and coccyx.

A somewhat different musculo-fascial technic was described by Mohardt and De Furia (1949)[16] and by Holman (1946)[12]. In their method, the sacrococcygeal fascia was incised and reflected to include part of the gluteal muscle and its fascia. Then these two reflected flaps were overlapped and sutured across the midline. They, too, were attempting to fill this natural dead space and gain a vascular pad at the base of the wound.

Recently, a few articles have appeared in disfavor of these musculo-fascial methods. The difficulty appeared to be in trying to approximate the lateral flaps over the sacrum. The tension was too great and some wounds broke down. In addition, they also noted recurrences at the distal end of the wound. They believed this was due to the anococcygeal raphe causing a deep dimple which eventually allowed hair to migrate into it and cause a new sinus tract. They mobilized the skin at the distal margin by separating it from the raphe in an attempt to prevent dimple formation. This complication will be discussed later when we describe our technic in treating pilonidal disease.

METHOD OF TREATMENT

From January 1951 to April 1953, we have had slightly over 500 patients admitted for pilonidal disease. Of this number 20 per cent were either returned to duty with no treatment or were separated from Service. Until September 1952, all patients operated upon were treated by the open method with an average

postoperative hospital stay of 80 days. In September 1952, a modification of the musculo-fascial procedure was suggested by one of the authors, (E. J. P.), and it was again decided to attempt closing the wounds. The method follows:

The patients are all prepared preoperatively with neomycin and sulfathalidine for 24 hours prior to operation. At 9 a.m., the day before operation, the patient is given 1 Gm. of neomycin and 1.5 Gm. of sulfathalidine each hour for three additional doses followed by the same dosage every four hours up to the time of operation. A purge of castor oil (60 cc.) is given with the first dose of neomycin and sulfathalidine. The patient is placed on a low residue diet.

Under spinal anesthesia, with the patient placed in the prone jackknife position (20°), the skin is prepared with septisol. The cyst is excised in one ellipitcal block of tissue extended down to the sacrococcygeal fascia. Hemostasis is obtained initially with warm packs followed by electric coagulation and an occasional suture-ligature for persistent bleeding points or by suture-ligatures of no. 000 plain catgut only. The deep subcutaneous fat then is reflected from the gluteal fascia. Longitudinal incisions are made into both gluteal muscles approximately 1.5 cm. deep and 2.5 to 3.0 cm. lateral to their attachment to the sacrum and coccyx. These medial musculo-fascial flaps are inverted and sutured in the midline over the sacrum and coccyx with no. 00 chromic catgut. At this point, care is taken to transect the anococcygeal raphe. This will obliterate the deep natal cleft proximal to the anus and allow the musculo-fascial flaps to fill this dead space. By this maneuver dimpling of the skin is prevented which frequently is the cause for the formation of a recurrent tract. The lateral cut edges of the gluteal muscles are allowed to retract without further surgical manipulation. The skin and subcutaneous layers are then closed by first placing a row of interrupted no. 00 plain catgut sutures in the deep portion of the subcutaneous layers, a second row of catgut sutures in the superficial portion of the subcutaneous layer and a running no. 0000 silk cuticular stitch in the skin. This cuticular stitch gives excellent skin approximation and comes away readily on about the sixth postoperative day.

Postoperatively, the patients are kept on constipating doses of bismuth and paregoric, a nonresidue diet and bed rest in the prone position for five days. Ambulation usually is completed by the tenth day and patients are sent to duty by the fourteenth to twenty-first day.

Of 125 patients whose wounds were closed with musculo-fascial flaps without drainage, 3 developed hematomas and were opened, and 2 required superficial re-excision and closure. Only 2 developed major sepsis; these were laid open and packed. When clean, after four to five days, the wounds were closed with through and through wire. It was noticed, however, that fluid collection in the depths of the wound was a frequent occurrence requiring multiple aspirations. In 6 patients in whom the skin wounds were too large to close, the medial muscle flaps were developed, sutured across the midline and the wounds packed open. Granulations from this muscular floor brought about rapid healing. The period of convalescence was 35 per cent less than when packed open without the use of muscle flaps.

The accumulation of fluid in the depths of the wound has indicated that the

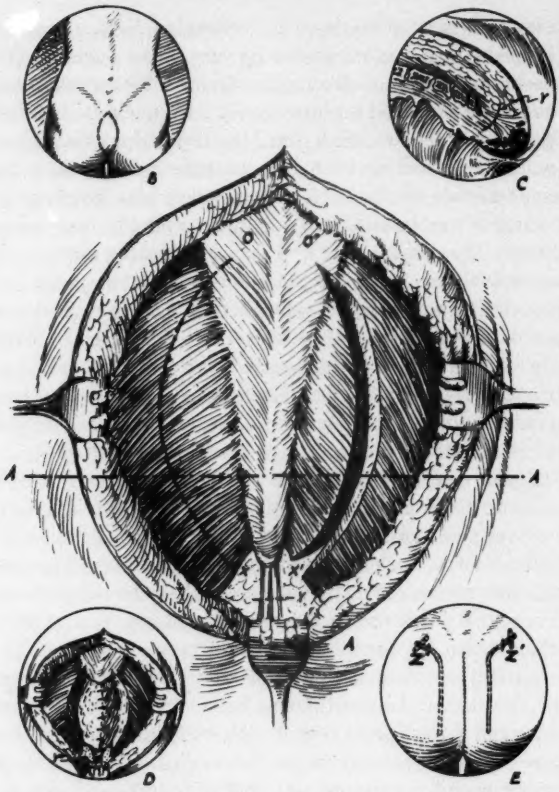

FIG. 1. A. The area exposed in the excision and undercutting of the skin and subcutaneous flaps. OX and O′X′ indicate the incisions into the glutei about 1.5 cm. deep into the muscles. At X and X′, the full thickness of the muscles is incised. X and X′ will be approximated when the muscle flaps are reflected to the midline. Y represents the fibrous strands to the midline raphe and sphincter ani muscles to be divided.

B. Lines of skin incisions.

C. Y indicates the level at which the strands of fibrous tissues extending from the coccyx to the sphincter ani and the midline raphe are divided.

D. OX and O′X′ have been reflected medially and sutured with a few interrupted stitches of catgut to cover the avascular sacral and coccygeal fasciae with a vascular muscle pedicle. Y indicates the division of the fibrous strands. Deep sutures are placed to obliterate the dead spaces by bringing in the fatty tissue from the sides and at the same time serving to obliterate the dependent portion of the natal fold.

E. The final skin closure with a cuticular suture of fine black silk. The deep subcutaneous fascia has been approximated with a few interrupted sutures of no. 000 plain catgut. Ordinarily, the tissues are not under tension. Z and Z′ show penrose drains along the depth of the incisions into the glutei muscles. These drains may be brought out through a single lateral stab wound if desired. They are left in place three or four days.

procedure should include the institution of drainage for the first three or four days postoperatively. Beginning in September 1953, the procedure was modified to include placing penrose drains along the depths of the incisions into the glutei bilaterally as shown in figure 1 E and figure 2 D. The use of drainage has proved

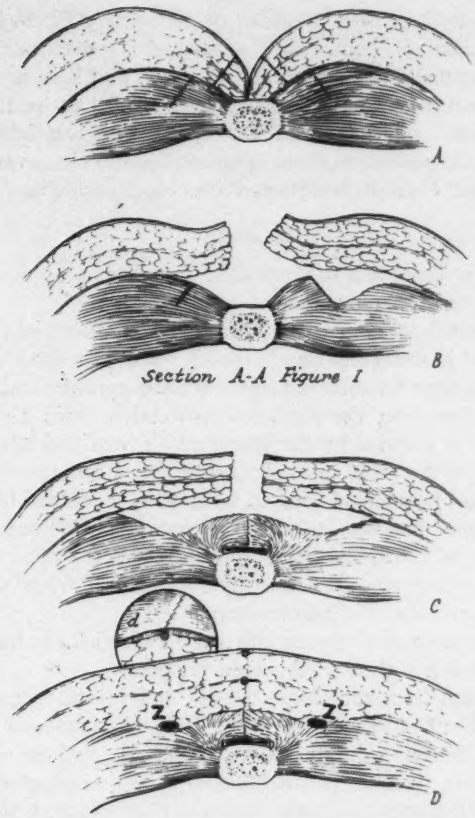

FIG. 2. Illustrating the excision, incision and closure at level AA in figure 1.

A. Showing the deep natal cleft, the block excision down to the sacral and coccygeal fasciae and the incision into the glutei. This incision into the muscles is about 1.5 cm. deep.

B. Showing the undercutting of the lateral skin and subcutaneous flaps and the retraction of the lateral edge of the glutei muscle when it is incised.

C. Showing the obliteration of the space over the sacral and coccygeal fasciae by the vascular medial muscle pedicles. The blood supply is primarily from the perforating branches of the presacral arteries.

D. Showing suture of glutei, deep and superficial subcutaneous fasciae with fine interrupted sutures of plain catgut. There is no tension. Z and Z′ indicate the locations of the two penrose drains placed in the depth of the incisions into the glutei.

d. Shows the cuticular stitch of fine black silk used to give accurate apposition of the epidermis. The medial muscle pedicles over the sacral and coccygeal fasciae obliterate the natal fold effectively, decrease the width of the defect and permit closure of the skin flaps without tension.

worthwhile in that wounds have healed more rapidly and have required much less expert attention. These drains must be placed well laterally and should never be brought through the midline wound. Drainage has been used in 28 consecutive patients and the improved results dictate continuation of the practice.

Recently patients have been prepared preoperatively for 20 hours with neomycin and sulfathalidine, (Poth, 1953[17]) as follows: (1) castor oil 60 cc., neomycin 1.0 Gm., and sulfathalidine 1.5 Gm. at 1:00 p.m., and then neomycin 1.0 Gm. and sulfathalidine 1.5 Gm. at 2:00 p.m., 3:00 p.m., 4:00 p.m., 8:00 p.m., 12 midnight, 4:00 a.m., and 8:00 a.m. The operation is scheduled at 9:00 a.m. Postoperatively, the patient receives sulfathalidine 1.5 Gm. every four hours, a low residue diet and bismuth and paregoric as constipating agents for five days.

SUMMARY

In summary, we would like to stress that we believe most recurrent cysts are due to improper closures or weak scars.

We have presented cases in which a gluteal musculofascial flap technic was used. Our rational in using this flap is that it helps fill a dead space; supplies a good vascular pad over the sacrum and coccyx and permits final closure without tension, thereby lessening the chance for defective scars. Even in the open wounds, recovery is speeded by the granulations from this muscular floor. We believe that it is undesirable to approximate the lateral muscle flaps, because then closure cannot be effected without tension. This method has enabled us to reduce postoperative hospital stay from an average of 80 days to one of 16.5 days. Breakdown of broad flat scars resulting from the open technic is not a problem. While primary closure ordinarily is possible, the basic procedure lends itself to use open treatment when necessary.

This technic gives excellent results and is recommended as a highly satisfactory procedure for dealing with a troublesome problem.

The authors wish to acknowledge the aid and helpful suggestions of many members of the Staff of the 3700th USAF Hospital, Lackland Air Force Base. Because of the magnitude and duration of this study and the changing hospital personnel, it is not possible to list all who have participated. However, the authors wish particularly to thank Captains Sam Mack, Charles R. Bowers, Charles G. Beachley, Jr., and Stanley M. Fromm.

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THE SURGICAL THERAPY OF ACUTE PERFORATED PEPTIC ULCER

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There is little doubt that the incidence of benign peptic ulceration of the stomach and duodenum is increasing. From all indications this increase is an absolute one and does not represent merely improved recognition of the problem associated with better diagnostic technic. Therapeutic programs of diet, ganglionic blocking agents and of psychotherapy are well established in myriad forms today and many patients are able to lead normal lives unhampered by the well-known symptomatology of peptic ulcers. A large group of patients. however, is plagued by a continuation of the annoying symptoms despite therapy, and others continue a symptomatic course because of unwillingness to follow the advice of their physicians. These patients are prone to develop one of the complications of gastric or duodenal ulceration such as hemorrhage, obstruction or perforation, although any of these may occur in the cooperative or asymptomatic individual. It is the purpose of the present study to relate the experience on the University Surgical Service of the Grace-New Haven Community Hospital in treating 191 patients with perforated ulcers during the years 1928 to 1953 inclusive.

Peptic ulcer perforation is an acute abdominal emergency which will eventuate fatally in many cases unless active intervention is forthcoming. Since the early days of the modern surgical era, specific therapy has consisted of suture of the intestinal defect in an attempt to halt the spreading peritonitis. There has been a high mortality rate, however, reported in some large operative series.[1, 3, 4, 5] This fact coupled with the observation that many patients with apparent perforated ulcers survive without surgical therapy has led to a reawakening of interest in a nonoperative or conservative method of management of this condition. Several British investigators[8, 9, 10] have reported in recent years groups of cases of patients who were treated conservatively with good results, while Seeley[7] has been a most active proponent of this form of therapy in the United States. Complete evaluation of the conservative approach has been difficult coming as it does at a time when the surgeon's knowledge of anesthesia, fluid and electrolyte balance, whole blood therapy, antibiotics and chemotherapeutic agents and modern surgical technic have achieved such a high degree of development. One is not justified in comparing mortality rates in a series of patients treated surgically several years ago with a series of patients treated conservatively today. Rather one must compare groups of patients treated surgically in different periods or groups treated surgically or conservatively during the same period. Only in this way can one decide which form of therapy, if indeed either, is preferable.

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TABLE 1

	1928–1940 (47 Cases)	1941–1953 (144 Cases)
Male	42	129
Age		
0–20 years	1	5
21–30	6	17
31–40	9	27
41–50	13	30
51–60	13	36
61 plus	5	29
Hours symptoms before operation		
0–6	27	48
7–12	10	50
13–24	4	21
24 plus	3	14
Previous ulcer symptoms		
Less than 1 month	6	13
1–6 months	2	12
7–12 months	5	8
1–2 years	3	6
2–10 years	13	46
10 years plus	7	24
None	10	31
Unknown	1	4
Location		
Duodenum	22	77
Stomach	12	32
Prepylorus	12	29
Unknown	1	6
Operation		
Plication	41	128
Gastrectomy	2	4
Excision ulcer	1	1
Gastroenterostomy	8	1
Conservative treatment	3	11
Roentgenogram (plain erect abdomen)		
Pneumoperitoneum	12	85
Indeterminate for pneumoperitoneum	6	39
No films taken	29	20
Time in hospital		
1–10 days	1	26
11–20 days	26	77
21 days plus	11	26
Deaths		
Operative	7	12
Conservative	2	3
Mortality rate (per cent)		
Operative	16	9
Conservative	67	27
Over-all	19	10

In the present series (table I) 177 patients were treated surgically and 14 patients were managed conservatively. To evaluate any improvement in therapy which might have taken place, the cases have been divided into two groups. Those patients from the years 1928 to 1940 inclusive have been placed in Group A (47 patients), while those treated from 1941 to 1953 inclusive have been designated as Group B (144 patients). The second group embraces the era during which modern drugs and technics have become commonplace. It should be noted here that only benign peptic ulcers have been studied. Perforated malignancies and acute ulcerations of the gastrointestinal tract in infants have been excluded.

Peptic ulcers are more common in the male population and in the present series there were 171 men. Age apparently does not play a part in the perforated ulcer syndrome, for even young people are affected. The age range in the present series was from 17 to 88 years, the greatest number of patients falling into the 40 to 60 year age group and 66 per cent of the patients being over 40 years of age.

Approximately one-half of the ulcers here could be located definitely in the duodenum, while one-quarter of them were gastric in origin. The remaining 25 per cent could be located only in the general region of the pylorus because of the great degree of inflammatory reaction which obscured the area at operation. In 7 of the patients treated nonoperatively, the site of the perforation was not determined. It is interesting to note that 12 patients had their perforations occur while they were in the hospital for various reasons, 8 of them undergoing upper gastrointestinal diagnostic studies.

The relationship of the duration of an ulcer to its perforation is not clear at this time. Forty-one patients (22 per cent) denied any previous symptoms suggestive of ulceration, while 90 patients (47 per cent) had symptoms or roentgenographically proved ulcers for over two years. Of this latter group, 31 (16 per cent) were symptomatic for over 10 years. Recurrent perforations were noted here in 6 patients. One patient had his first prepyloric perforation at age 60 and recovered uneventfully following plication. Gastrectomy was refused. Eighteen months later his ulcer reperforated at the same site and again was plicated. A definitive procedure was refused a second time. Nineteen months after the second episode the patient again was admitted with signs and symptoms of perforation and was treated expectantly at this time with quick recovery.

The diagnosis of a perforated peptic ulcer usually is not a difficult one to make because of the usual past history of a proved ulcer or a history of ulcer symptomatology; the onset of incapacitating sharp, abdominal pain; and the finding of signs of peritoneal irritation on physical examination. An erect survey roentgenogram of the abdomen is helpful in that free air commonly is seen beneath the diaphragm. Instillation of diodrast into the upper gastrointestinal tract via a gastric tube with roentgenograms afterward has been suggested as one way to determine whether a suspected perforated ulcer has been walled off or is still patent.[6] In the present series two-thirds of the plain films taken showed free subdiaphragmatic air. Of the patients treated conservatively pneumoperitoneum was demonstrated in 80 per cent of the films taken. All patients with one exception in the conservatively treated group, whose roentgenograms did not reveal

free intraperitoneal air, had perforated peptic ulcers demonstrated at postmortem examination. This exception was a 58 year old man who had a duodenal ulcer diagnosed by gastroduodenal contrast roentgenograms four years prior to admission. One previous episode of perforation with pneumoperitoneum seen on a roentgenogram had been treated expectantly at another hospital two years before. The patient was admitted to the University Service with a history of five hours of abdominal pain and physical findings compatible with a perforated hollow viscus. Surgery was refused and conservative management was instituted. The patient was discharged asymptomatic 13 days after admission.

Patients with perforated peptic ulcers in most cases die of rapidly spreading, overwhelming peritoneal inflammation. The ultimate mortality rate is dependent upon the duration of the postperforation peritonitis. Six hours has been established by past experience as being the critical time after perforation during which therapy must be started if a patient is to be given the best opportunity for recovery. The present series of patients included 39 per cent whose perforations probably occurred less than six hours before specific therapy was instituted.

Since the present series was not planned as others[2] to evaluate forms of therapy, the surgically treated patients in Group B were patients actually treated by a combination of conservative and surgical methods. Customary treatment in this hospital during the past 10 years includes constant gastric suction, fluid replacement and blood administration, when indicated, as well as the use of chemotherapeutic and antibiotic agents preoperatively in suspected cases of perforation. All of these measures, therefore, were used to some extent in the Group B surgical patients while most of the measures were not used in the Group A cases. For example, antibiotics and chemotherapeutic agents were administered to 64 per cent of the Group B patients either singly or in combination, while none was administered to patients in Group A.

The patient who has a perforated hollow viscus is critically ill because of the local pathology as well as the general body reaction to it. Any operative procedure should be done under anesthesia of the shortest duration possible. Spinal anesthesia is perhaps somewhat safer than a general agent and was used in slightly less than one-half of the present group, its use being somewhat greater in Group B than in Group A patients. Simple plication of the perforation with an omental tab incorporated in the closure is perhaps the best emergency surgical precedure and such was done in 89 per cent of the cases. Gastrectomy obviously involves more trauma to the patient and was done in only 6 patients in this series. All these patients were in good general condition for such a procedure. Local excision of the perforation and associated ulcer was considered to be indicated by the surgeon in 2 patients because of associated massive hemorrhage. Symptomatic pyloric obstruction was noted in the admission histories of 9 patients and was confirmed at operation. In these patients gastroenterostomy was done as well as plication of the ulcer at the initial operation. Eight of these patients were operated upon before 1940.

There were seven postoperative deaths in Group A (16 per cent). Five of these patients never rallied completely after simple plication of the perforation in all patients, and gastroenterostomy in addition in 2 patients. Another patient died

of massive upper gastrointestinal hemorrhage 14 days postplication of a gastric perforation, and at postmortem examination was found to have ulceration of the gastroduodenal artery. The remaining death was in a man who developed a pelvic abscess after plication. He died suddenly while under anesthesia as the abscess was about to be drained.

In Group B there were 12 postoperative deaths (9 per cent). Four patients progressively deteriorated after plication and died in the immediate postoperative period despite vigorous therapeutic measures. Another patient developed pyloric obstruction after plication and died following an emergency gastroenterostomy. Three other patients developed peripheral vascular collapse following plication of their perforations and died shortly thereafter. A cerebrovascular accident caused the death of 1 patient seven days after plication and another patient had cardiac irregularities four days after operation and subsequently died. The eleventh patient perforated while in the hospital for gastrointestinal studies. At operation it was thought that a gastric carcinoma had perforated and simple plication was done. No tissue for biopsy was taken. The patient was later re-explored and was then considered inoperable. A downhill course to death followed and at postmortem examination only a large walled-off abscess involving the posterior stomach, pancreas and mesentery could be found. The final death was that of a man who was admitted in shock from upper gastrointestinal hemorrhage and probable perforation. At operation a perforated duodenal ulcer was plicated, but the patient died during the wound closure.

Conservative therapy was instituted in the present group either because the patient refused operation following the acute episode or because it was believed by the attending staff that the patient's poor condition precluded surgical intervention. In Group A only 3 patients were treated nonsurgically and 2 of these were moribund on admission having perforated 24 and 96 hours respectively before admission. Both of these patients subsequently died (67 per cent). The remaining patient refused operation and recovered following expectant management.

Eleven patients in Group B were treated conservatively, including 3 patients who perforated while in the hospital, and died a short time later (27 per cent). The remaining 8 patients had uneventful recoveries.

There was little difference in the length of hospitalization in each group of surgically treated patients; 61 per cent of the total discharged remaining from 11 to 20 days. The conservative group who survived had an average hospital stay of nine days. Postoperative complications were few in the total operative group and included 4 pelvic abscesses, 3 subhepatic abscesses, 3 wound dehiscences, 3 subdiaphragmatic abscesses and 1 gastric fistula. Nine pulmonary complications developed, including 5 cases of atelectasis, 3 cases of pneumonitis and 1 pulmonary infarct. All complications were treated adequately and the patients were well on discharge from the hospital.

COMMENT

Much has been written in recent years regarding the relative advantages of treating peptic ulcers which perforate by either surgical or nonsurgical methods.

Mortality rates differ sharply from study to study and it becomes apparent that there really is no *best* method of therapy. Rather each patient must be individualized in terms of duration of the perforation and general physical condition before any therapeutic program is initiated. Of special importance is any evidence of localization of the intraperitoneal inflammatory process. If a young patient is seen in the early stages of the disease; is in good condition physically and localizing signs are absent, surgical therapy probably is best. It should be added, however, that the surgically treated patient should have the obvious benefits of gastric suction, antibiotics or chemotherapeutic agents and parenteral fluids or blood as indicated prior to surgery. On the other hand, the elderly, malnourished patient with a perforation that is 24 hours old probably would benefit more from a conservative regime. The shock of operation and anesthesia, however brief, could be fatal in such a patient. It cannot be too strongly emphasized that these case examples are extremes and each case must be evaluated clinically on its own merit. Therapy should be based on this evaluation.

The present series is reported as an analysis of the surgical approach to the problem in a large number of cases. It is significant that the postoperative mortality rate decreased from 16 per cent in 1928 to 1940 to 9 per cent in the period 1941 to 1953. This decrease obviously represents the benefits derived from the advances made in medicine during the latter era. The number of patients treated conservatively was too few for any conclusion to be drawn regarding the value of such therapy. When these patients are included, the over-all mortality rate for the period 1928 to 1940 was 19 per cent while the mortality rate was 10 per cent for the period 1941 to 1953.

CONCLUSIONS

The mortality rate in 44 patients with perforated benign peptic ulcer treated surgically from 1928 to 1940 was 16 per cent, while the mortality rate in 133 patients treated surgically from 1941 to 1953 was only 9 per cent.

Every case of perforated peptic ulcer must be individualized regarding the decision to employ surgical or nonsurgical methods of therapy.

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SPHINCTER PRESERVING OPERATIVE PROCEDURES FOR CANCER OF THE RECTUM, RECTOSIGMOID AND LOWER SIGMOID*

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Efforts to cure carcinoma of the rectum, rectosigmoid and lower sigmoid have varied from local circular excision of a lesion on the rectal wall through a posterior incision to the combined abdominal and posterior approach removing the entire left colon, rectum and anus. All early operative efforts in cancer of the rectum were directed toward preservation of the sphincter, including the earliest report of Lisfranc[22] in 1826, through a posterior approach, a tumor of the rectal wall was excised by a circular incision and the defect in the bowel wall closed. At intervals, case reports were made of various types of local excision and then in 1885 Kraske[21] gave a new impetus by describing his more radical technic. After excision of the coccyx and a segment of the sacrum, the lower, mid and upper rectum could be removed and bowel continuity re-established by an end to end anastomosis. By 1889 Hochenegg[19] had reported his "*pull through*" procedure which consisted of freeing the lower colon and rectum through a posterior approach and invaginating the tumor-containing segment out through the dilated anus. Here the protruding segment was amputated and the bowel sutured to the anal margin. This technic lessened infection and fistula complications. A more radical procedure was presented by Maunsell[24] in 1892 and could be extended to lesions of the lower sigmoid. He introduced the combined approach. The sigmoid and rectum were freed by abdominal dissection and then the tumor was intussuscepted through the split anal sphincter and anastomosis accomplished posteriorly, similar to the Hochenegg technic. Weir[31] and others presented a modification of this technic during the next few years.

The Miles[26] era began in 1908 when he modified the entire objective for operating upon cancer of the rectum and terminal pelvic colon. He minimized the necessity for retaining the sphincter mechanism and emphasized the desirability for a radical removal of all contiguous tissue and lymphatics, including the anal sphincter. His premise was based upon the studies of the lymphatics of the rectum and rectosigmoid by Poirier, Cuneo and Delamere[27] from which he concluded that cancer in this region extends upward, laterally and downward. This combined abdominoperineal procedure required a permanent abdominal colostomy. To fortify the conscience of the patient and surgeon there developed propaganda about the compatibility of an abdominal colostomy with continued social, economic and athletic activities. Time has proved that this Miles operation in one or two stages has resulted in the highest percentage of cures for carcinoma of the rectum, rectosigmoid and lower sigmoid. However, the stigma

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of an abdominal anus could not be forgotten even though thousands and thousands of patients lived and are living an active life with a colostomy. During these last 45 years, the abdominoperineal excision rightly has been widely accepted, but nevertheless there have been sporadic attempts to develop operative procedures that would sufficiently answer the need for wide excision and yet preserve anal continence. Lockhart-Mummery[23] in 1908, and Balfour[4] in 1910 reported their experiences with lesions of the lower sigmoid and rectosigmoid with anastomosis over a rectal tube. Interest was further stimulated by the reports of Babcock[2] in 1932 and Bacon[3] in 1938 on a modified *"pull through"* operation somewhat similar to the technic of Maunsell. The *"pull through"* operation has not been widely accepted because sphincter control is too frequently inadequate in the experience of many surgeons. Also, in our opinion, the operation does not appear to be sufficiently radical in lesions of the mid-rectum because the levator ani area cannot be so widely excised through the anal approach. Since 1935 Devine,[11] Horsley,[20] Dixon,[12] Fallis,[13] Wangensteen,[29] Waugh and Kirklin,[30] Garlock and Ginzburg,[15] Quer, Dahlin and Mayo,[28] Welch and Rheinlander[32] and d'Avilo[1] among others, have presented operative technics for preservation of the sphincter mechanism.

Previous to 1941, we had occasionally restored intestinal continuity for lesions of the lower sigmoid, but never for lesions involving the rectosigmoid. In 1941, after reviewing the literature to that date on the involvement of lymph nodes in carcinoma of the rectum, we were impressed by the infrequency of metastatic lesions and the minimal intramural extension of the disease below the lower palpable margin of the tumor. Also these reports related minimal involvement of paracolic nodes to any great distance above the tumor unless there was

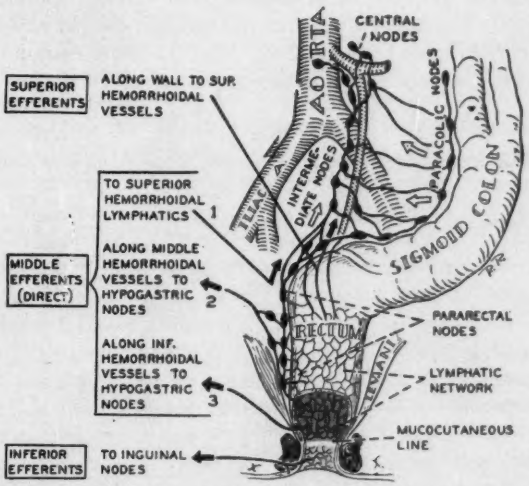

FIG. 1. Diagram of the lymphatic pattern for the anus, rectum and rectosigmoid. The usual dissemination of cancer cells is toward the intermediate and central group of lymph nodes. (From Ann. Surg., Sept., 1949)

blocking metastasis to the intermediate and central nodes (fig. 1). Although we accepted the infrequency of downward spread of the disease, we could not quite accept the remarks on the degree of upward dissemination at that time. Therefore, we proposed to transect the rectum at least 2½ cm. below the lower margin of the rectal lesion, remove the left colon in a radical manner and then anastomose the ileum to the rectal stump. We[6] were not happy with these ileorectostomy cases because of fistula complications and the incidence of diarrhea and we discontinued this procedure for carcinoma of the rectum and rectosigmoid. It was also becoming apparent that probably it was not so necessary to be so radical in removal of the upper left colon since the paracolic nodes were so infrequently involved, unless the intermediate and central group of nodes were involved.

There is sufficient material in the literature to attract one's attention to the infrequency of the extrarectal and intramural retrograde extension of carcinoma of the lower sigmoid, rectosigmoid and rectum to warrant a re-evaluation of the abdominoperineal resection and permanent abdominal colostomy. We have collected 607 cases from the literature where special studies were made of collected specimens from operations or autopsies for extrarectal lymph gland and intramural spread of carcinoma. Only 5 cases, an incidence of less than 1 per cent, revealed retrograde spread beyond 2 cm. (table I). Unfortunately too little attention was given to the report of McVay[25] in 1922 on the involvement of lymph nodes in 100 cases of carcinoma of the rectum as he reported only one node was involved below the lower margin of the lesion and this was at the 1 cm. level. These cases are included along with those studied later by Wood and Wilkie,[34] Westhues,[33] Gabriel, Dukes and Bussey,[14] Gilchrist and David,[16] Coller, Kay and MacIntyre[10], Grinnell[18] and Glover and Waugh.[17] More recently Quer, Dahlin and Mayo[28] have studied 89 fresh specimens from abdominoperineal resections in patients where it was believed a curative condition existed and only one had intramural retrograde spread beyond 2 cm. from the lower margin of the tumor. With these statistics, we are unable to be complacent about the last word having

TABLE I

Collected data on incidence of lymph node involvement below margins of malignant lesions

Author	Year	Cases Studied	Cases With Nodes (1 to 2 cm. Below Lesion)	Cases With Nodes (2 cm. or More Below Lesion)
McVay	1922	100	1	0
Wood and Wilkie	1933	100	0	0
Westhues	1934	74	1	0
Gabriel, Dukes and Bussey	1935	100	2	0
Gilchrist and David	1938	25	0	2
Coller, Kay and MacIntyre	1940	33	1	0
Grinnell	1942	75	1	0
Glover and Waugh	1944	100	6	3
Total cases		607	12	5
Percentage			2.3	.82

been said about the treatment of carcinoma of the lower sigmoid, rectosigmoid and rectum and that routinely in every case eradication of the sphincter mechanism is indicated. It has become necessary to select or design operative procedures which would appear to be sufficiently radical and yet leave the patient with a continent anal sphincter. All early operative procedures by the posterior approach had to be discarded, including the Kraske and Hochenegg operations. The Maunsell operation with the abdominal dissection and the "*pull through*" procedure by the posterior anal approach was not satisfactory because frequently the sphincter mechanism was not adequate and the operation was not sufficiently radical in the levator ani area for the mid rectal lesions. In our opinion this also holds true for the modified "*pull through*" procedure of Babcock and Bacon. It did seem logical that for lesions of the lower sigmoid and many of those of the rectosigmoid, a radical abdominal dissection and a wide pelvic dissection extending below the peritoneal pelvic floor as in the abdominoperineal resection would meet the requirements of a good cancer operation, provided one transected the rectum at least 2½ cm., and preferably 5 cm., below the lower margin of the palpable tumor. In the earlier group of cases, we extended this abdominal operation to lesions within 5 cm. or less of the anal sphincter. After further analysis of anatomic studies of the levator area and our own injection studies[7, 9] of the lymphatics of the area, it was evident that the abdominal operation could not be sufficiently radical to remove the lateral zones of spread in lesions below the 10 cm. level or below the peritoneal pelvic floor. Lesions in this area and below would be adjacent to the levator ani structures and the group of middle efferent lymphatics which course above and below the levator muscle and fascia and lead to lymph nodes along the internal pudendal vessels and the hypogastric nodes along the internal iliac vessels. Therefore an adequate operation for lesions at the 10 cm. level or lower requires an abdominal dissection and a posterior excision of the levator muscles, fascia and adjacent structures. This can only be accomplished satisfactorily by a combined abdominal and radical posterior approach. The question then arises as to where the line should be drawn in attempting to preserve the sphincter mechanism in low lying rectal lesions. Since one should go at least 2½ cm., and preferably 5 cm., below the lower margin of lesions in the upper rectum, we did not believe that the margin of safety should be narrowed in these low lying rectal lesions. Therefore, it is our opinion at the present time that lesions which are 5 cm. or less from the external anal margin require an abdominoperineal procedure with sacrifice of the sphincter mechanism.

Based on our experiences with over 150 patients, a few technical points should be of interest.[5, 7, 8] The rectosigmoidectomy no. 1 procedure, which is abdominal dissection, resection and anastomosis, is indicated for lesions of the lower sigmoid, rectosigmoid and lesions of the upper rectum which are not entirely below the peritoneal pelvic floor and are 10 cm. or more above the external sphincter margin. Paramedian, transverse and oblique incisions have been used. A transverse incision an inch or so below the umbilicus, transecting both recti muscles and with the incision flaring upward on the left, has been satisfactory for freeing the left colon and splenic flexure as necessary and for working deep in the pelvis.

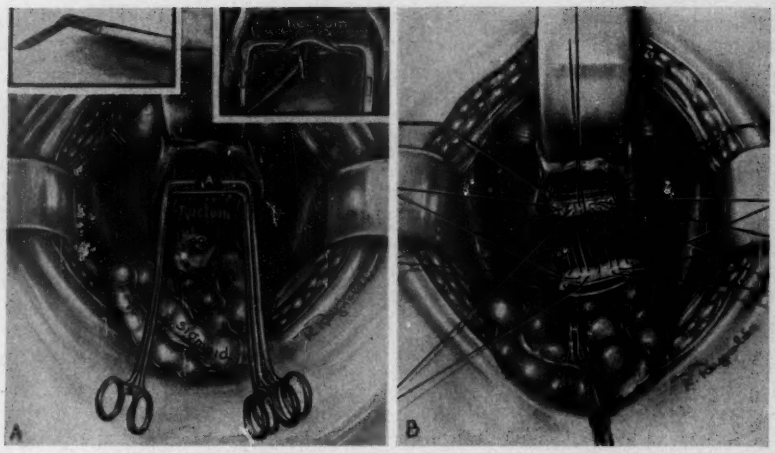

FIG. 2. A. The special clamps are applied to keep leakage at a minimum and to steady the rectal stump during the transection in the Rectosigmoidectomy no. 1 procedure. B. The shorter angle clamps are removed and replaced by a guy suture on each side. Interrupted silk guide sutures are inserted to permit sliding the upper colon segment onto the rectal stump.

The superior hemorrhoidal vessel is ligated just below the left colic branch of the inferior mesenteric and on occasions we ligate the inferior mesenteric above the left colic branch, but always preserving the marginal vessels of the left colon. A special set of right angle clamps, fairly heavy but not crushing in type, have been found most advantageous in securing the rectum in the hollow of the sacrum

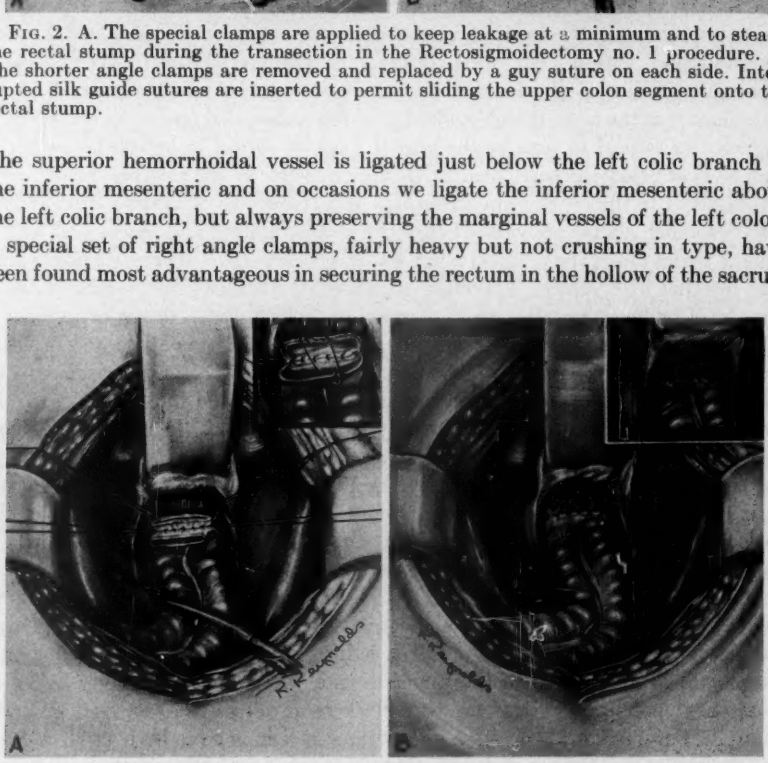

FIG. 3. A. The posterior guide sutures have been tied and a row of interrupted catgut sutures have been placed and tied. This layer of catgut sutures is continued around onto the anterior wall with knots on the mucosal surface. B. A layer of interrupted figure of eight silk sutures is placed anteriorly.

and steadying it for the correct level of transection. The anastomosis is accomplished as depicted in figures 2 and 3. Since we go low in the space in the hollow of the sacrum, a rubber drain is placed here and at the conclusion of the operation a small incision is made alongside the coccyx and the drain is delivered. This is true dependent drainage. The peritoneal floor is closed above the suture line. A cecostomy is done using a no. 34 or 36 mushroom catheter, bringing the tube out through a small stab wound incision and securing the cecum to the parietal peritoneum. This serves as a decompressive measure and usually a nasogastric tube is not necessary. If leakage occurs at the site of anastomosis, there usually is sufficient sidetracking of colon contents to permit healing of the fistulous tract. This cecostomy tube is irrigated night and morning with an ounce of tap water. About the seventh or eighth day, increasing amounts of water are instilled and the tube is clamped most of the time to encourage colon evacuation. After several bowel movements have occurred, the tube can be removed and the cecostomy site will close. A rectal examination is done at the end of two weeks, and if the site of anastomosis is palpable, one makes sure that too much narrowing has not taken place. If the area admits the index finger easily, evacuation function has seemed to be adequate.

The rectosigmoidectomy no. 2 procedure is abdominal dissection followed by posterior resection and anastomosis. It is indicated in lesions less than 10 cm. and yet more than 5 cm. from the external anal sphinster margin and for most lesions that are below the peritoneal pelvic floor level. The abdominal and pelvic dissection is carried out as for an abdominoperineal resection. It is important to free the left colon and usually the splenic flexure in order to have sufficient bowel length for the posterior anastomosis. A heavy silk ligature is tied around the

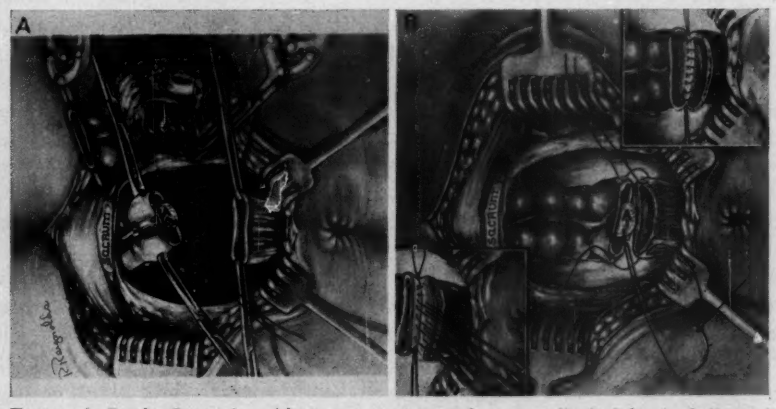

FIG. 4. A. In the Rectosigmoidectomy no. 2 procedure, a radical abdominal dissection is accomplished through an abdominal incision. The patient is turned onto the left side and the coccyx removed for the posterior radical resection. A larger segment of bowel is removed than depicted. A gauze pledget (A) prevents leakage while sutures are being placed. B. The angle sutures are placed and a complete row of anterior interrupted silk muscular sutures inserted before tying. The mucosal margins are approximated by a layer of interrupted chromic catgut sutures.

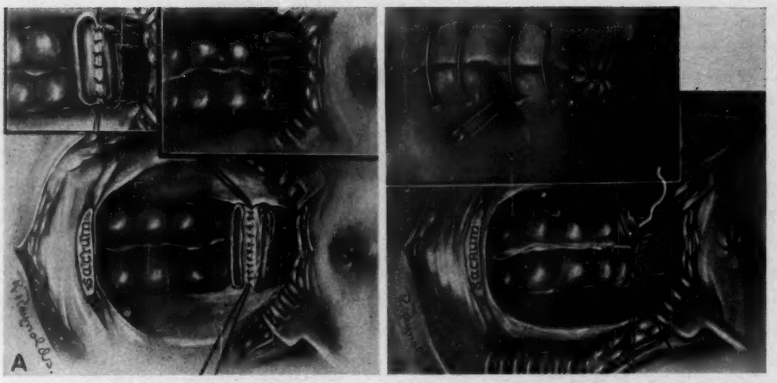

FIG. 5. A. After completing the layer of interrupted catgut sutures to the mucosa with knots on the lumen side, the interrupted figure of eight sutures are placed on the posterior muscular wall. B. After completing the interrupted posterior silk sutures, a drain is placed in the hollow of the sacrum and the skin closed.

colon at the proposed site of the anastomosis. The vessels have been ligated and in the interval one can identify the level of viable left colon. The peritoneum at the pelvic floor level is closed loosely around the sigmoid where it enters the true pelvis. In our early reports we stated that a cecostomy appeared to be adequate for the group of rectosigmoidectomy no 2 patients. However, further experience proved this was a misstatement as too frequently an annoying posterior fistula developed which required a later transverse colostomy before healing took place. Then a third operation was necessary to close the colostomy. After a few of these bitter experiences, it was apparent to us that where one made the anastomosis through a wide posterior approach, a temporary colostomy of the transverse colon was imperative. After closure of the abdomen, the patient is turned onto the left side, the coccyx is removed and the bowel delivered. The silk ligature identifies the level of transection. Anastomosis is accomplished as in figures 4 and 5. The colostomy is closed 8 to 10 weeks later. In the interval the site of anastomosis is dilated every 6 to 10 days, or as necessary. A channel which admits the index finger has been found to be adequate. In many of these rectosigmoidectomy no. 2 patients, the left colon is anastomosed directly to or within 1 inch of the anal sphincter structures.

Sphincter control has been adequate in all of the patients operated upon. Sexual impotence has been present in only about 20 per cent of the patients as against as least 90 per cent in the abdominoperineal resection group. A survey is now being made of over 150 patients and the preliminary figures reveal about the same percentage of recurrence rate and similar figures for three and five year survival rates as for abdominoperineal resection. We have been able to salvage several patients with recurrence—at least temporarily—by doing an abdominoperineal resection at a later date. When recurrence does take place, the onset of symptoms and the rectal and proctoscopic recheck examinations reveal the tumor, thus permitting one to attempt a secondary operation. In patients with

abdominoperineal resections, when recurrence is demonstrable it is usually too far advanced for further surgery except in the occasional patient with only a tumor deposit along the scar of the posterior incision.

SUMMARY

A critical analysis of the studies on the downward spread by extrarectal lymph node metastasis and intramural bowel wall extension reveals a less than 1 per cent incidence beyond 2 cm.

Greater emphasis must be directed toward more radical excision of tissue laterally in lesions of the rectum and particularly those between the 5 and 10 cm. level or below the peritoneal level.

For lesions of the lower sigmoid and rectosigmoid and upper rectum which are not entirely below the peritoneal level, the rectosigmoidectomy no. 1 operation with abdominal dissection, resection and anastomosis is recommended. A supplemental cecostomy is done.

For lesions between the 5 and 10 cm. level and usually those entirely below the peritoneal pelvic floor, the resosigmoidectomy no. 2 procedure of abdominal dissection but with posterior resection and anastomosis is recommended. Here a temporary colostomy of the transverse colon is indicated.

The abdominoperineal resection is recommended for lesions 5 cm. or less from the external anal sphincter margin.

At this time, it appears that one can depend on adequate sphincter function and the sexual impotence incidence is not nearly as high as with abdominoperineal resection. A preliminary survey of patients today reveals about the same incidence of recurrence rate and three and five-year survival rates.

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SURGICAL TREATMENT OF GASTRIC DIVERTICULA*

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Although relatively infrequent, gastric diverticula have been recognized and described for over 180 years. The reported incidence varies from 0.015 per cent to .3 per cent based upon roentgenologic and gastroscopic studies, and surgical and necropsy specimens. There apparently is no significant sexual or racial predilection. Diverticula can occur from infancy to the eighth decade, but most commonly are discovered during the fourth and fifth decades. Michel and Williams[2] stated that about 65 per cent of all gastric diverticula are found near the lesser curvature on the posterior wall of the cardia of the stomach.

The currently accepted classification of these diverticula is: 1. True (congenital —all the layers of the gastric wall are present without definite evidence that organic disease played an etiologic role. 2. False (acquired)—all layers are present with thinning of the layers and with evidence that some disease was an etiologic factor. The latter are further divided into, (a) pulsion—resulting from intragastric pressure, and (b) traction—secondary to extragastric adhesions.

Among the various etiologic causes suggested are congenital muscular weakness of the gastric wall, prolonged elevation of intragastric pressure, foreign bodies, pathologic conditions of the gastric wall, perigastric inflammatory adhesions, hernial formations of mucosa through vascular orifices and intragastric trauma. Pulsion and traction have roles in the etiology. According to Casberg and Martin,[1] if certain points of neural weakness are associated with conditions in which there is increased intragastric tension or extragastric adhesive traction, then the stage is set for possible development of a diverticulum.

Diverticula are almost always single but can be multiple or multiloculated. Essentially, a gastric diverticulum is an evagination of the stomach wall. It is seen as a small, rounded, sharply defined pouch protruding from the gastric lumen. The pouch may consist of full thickness gastric layers or these may be thinned out. It may measure from a few millimeters to several centimeters in length. The mouth may be broad or narrow, and the sac round, oval or pear shaped. The size of the orifice varies greatly. Diverticula with small orifices tend to retain gastric contents. With variations in filling, the sac changes in contour and may show an air fluid level. Some mobility usually is observed and the region, if accessible to palpation, is nontender.

The underlying causes cannot often be distinguished by roentgen examination but a practical classification of two main groups may be employed: (1) diverticula occurring in the juxtacardiac position, probably related to congenital weakness and seldom giving rise to complications (the majority of reported cases fall in this group); (2) diverticula occurring elsewhere in the stomach, usually associated with other disease.

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The diagnosis of a diverticulum of the stomach usually is made by roentgenogram in the process of searching for the cause of vague symptoms of epigastric distress. A clinical diagnosis prior to roentgenologic study or operation rarely is made. A clinical diagnosis has been recorded once in Tracey and Colcock's[3] report of a series seen in the Lahey Clinic. Complete aspiration of the gastric contents should precede roentgenologic study.

The roentgen criteria to be observed in establishing diagnosis are: (a) demonstration of rugae in mucosal lining of the sac; (b) a smooth, defined and regular shadow (except in the presence of diverticular gastritis or ulceration); (c) demonstration from several angles; (d) usually no tenderness elicited over the area of filling; (e) characteristic location at the cardia posteriorly; (f) retention of barium in the pouch for 6 to 24 hours; (g) occasional ability of the sac to empty with change in position; and (h) a possible gas and fluid level.

We advocate gastroscopy as an aid to roentgenologic diagnosis. It aids mainly in eliminating other diseases and should be done on all patients preceding surgery. However, gastroscopy frequently fails to locate a diverticulum demonstrated by roentgenogram so that it may be limited in value. When seen by a gastroscope the orifice of a true diverticulum has the appearance of a circular hole, the margins of which are well rounded and sharply defined. The surrounding mucosa usually is normal and there is no sign of infiltration. It may be added that gastroscopic observations of diverticula of the stomach are infrequent in the literature.

No other portion of the digestive tract, with the exception of the jejunum-ileum region, is so rarely the site of a diverticulum. The incidence of gastric, diverticula as compared to the remainder of the alimentary tract is as follows, with decreasing frequency: (1) colon, (2) rectum, (3) duodenum; (4) pharynx; (5) esophagus; (6) stomach; and (7) jejunum-ileum.

Gastric diverticula merit attention as sources of confusion with other pathologic changes in the stomach and as possible causes of obscure abdominal complaints. The symptoms are in no way characteristic. There is no syndrome pattern which fits this clinical entity. A large percentage of patients are asymptomatic so that one must not attribute existing symptoms to such a condition, unless other possible gastrointestinal disorders have been ruled out.

The most common complaints are: epigastric discomfort, variably affected by food or alkali; gaseous eructations; nausea; vomiting (especially old food particles); pain (upper abdominal or epigastric in location which may be dull, aching or burning); tenderness in epigastrium; dysphagia; sensation of epigastric pressure or fullness; nocturnal epigastric distress; malaise, relieved by vomiting; hematemesis; lower chest pain; weight loss; anorexia; and weakness. The symptoms depend upon the size, site, food retention and the existence of associated diseases.

In the differential diagnosis the following conditions should be considered: ulceration of the distal end of the esophagus; hiatus hernia; cascade stomach (dependent mobile gastric cardia); benign penetrating gastric ulcer; malignant gastric ulcer; diverticulum of duodenum or jejunum overlying stomach; and epiphrenic esophageal diverticulum. The serious complications of perforation and hemorrhage, although existent, are extremely rare. The most frequent com-

plications are retention with distention and inflammation. Tumors within diverticula have been reported.

The most commonly reported conditions associated with gastric diverticula are (1) gastric ulcer, (2) duodenal ulcer, (3) chronic gastritis and (4) gastric tumor.

We believe asymptomatic diverticula require no treatment. Mildly symptomatic diverticula should be treated medically for a reasonable period of time by frequent small feedings of a convalescent ulcer type diet, postural drainage and antispasmodics. Definitive surgery is recommended for diverticula with marked symptoms with definite retention, of large size or showing complications. With modern surgical technics the operation presents no great difficulty.

Our preference for surgical treatment is excision of the diverticulum by the left transthoracic approach. In the past, the lasting benefits of surgical removal have been denied many patients because of the technical difficulties associated with excision via the abdominal approach. Associated adhesions and increased vascularity in the region of the diverticulum, together with its high location on the posterior wall of the stomach, have made removal and safe closure of the remaining gastric defect almost impossible in many cases when attempted abdominally. With our present understanding of thoracic physiology and the great advances made in anesthesia recently, the transthoracic approach makes removal a safe and relatively simple procedure.

TECHNIC

The patient is anesthetized and an endotracheal tube is inserted. Anesthesia with cyclopropane, helilum and small quantities of ether, which can be administered with a high proportion of oxygen, is preferred. The patient is then placed in the left lateral position on the operating table and suitably braced so as to maintain this position. The skin incision is made over the left eighth or ninth rib from the midaxillary line to the paravertebral region and then extended superiorly so as to fashion a flap. The eighth rib is removed subperiosteally and the pleura is entered through the bed of the rib. After opening the pleura the associated intercostal vascular bundle is ligated and divided. Usually 1 inch segments of the rib above and below the removed rib are also excised, after which the wound can be widely separated. The base of the left lung is then separated from the diaphragm, and the pulmonary ligament is divided. This latter step aids in retracting the lung superiorly from the diaphragm. The diaphragm is then incised in a radial manner, directing the incision toward the esophageal hiatus. Bleeding from the divided edges of the diaphragm is controlled by transfixion sutures of silk which are left long to serve as retractors for the diaphragmatic opening.

On opening the diaphragm, the spleen and fundus of the stomach will be apparent. The former is pushed inferiorly and the stomach is grasped with Babcock forceps. Traction on the stomach demonstrates the gastrolienal ligament which is divided. There usually are several large vessels here which easily and safely can be clamped, divided, and ligated. This provides an opening into the lesser omental bursa and the position of the diverticulum now can be visualized. Usually it is

adherent posteriorly but can be mobilized safely and adequately under direct vision. It may be necessary at this point to ligate a few large vessels coursing over the diverticulum. With traction on the fundus of the diverticulum, the neck is demonstrated and traction sutures of silk are placed, outlining the limits of resection. The area is then packed off with moist sponges and, with a suction apparatus available, the diverticulum is excised, leaving a cuff ½ centimeter wide or less. Clamps are not used on the stomach. The opening in the stomach is closed with a continuous suture of fine catgut. We usually place a second continuous catgut stitch over the first to invert the suture line and provide serosa to serosa approximation. Interrupted silk Lembert sutures complete the gastric closure.

The stomach is returned to its natural position and the diaphragm is closed with interrupted sutures of nonabsorbable material. We have not found it necessary to drain the subphrenic space, but this may be advisable if there has been excessive spillage. The ribs are approximated with interrupted catgut sutures and the chest muscles with interrupted sutures of silk or cotton. A Pezar catheter is placed in the costophrenic angle, brought out through a stab wound and attached to a water seal with negative pressure to aid pulmonary re-expansion. We usually do not crush the phrenic nerve.

Postoperatively, the patient is given prophylactic wide range spectrum antibiotics, and deep breathing and coughing are encouraged. Nasal suction with a gastric tube is continued until peristalsis is active to avoid gastric dilatation. The fluid balance is maintained by intravenous fluids during this period. These patients are then treated as usual partial gastrectomy cases. Negative pressure is continued in the pleural space for 48 hours, after which pulmonary re-expansion is checked by a portable chest roentgenogram and, if adequate, the Pezar catheter is removed. The patient is allowed out of bed on the third postoperative day and discharged about the tenth.

SUMMARY

The rare occurrence of gastric diverticula is noted.

There is no typical syndrome which describes the clinical entity of gastric diverticula.

The difficulties of diagnosis and differential diagnosis are discussed.

Although therapy understandably can begin with a medical program, definitive treatment offering complete cure is surgical excision.

Surgery in these cases should be advised more frequently as the postoperative complications are minimal and recurrences of the diverticula are improbable.

A technic for the removal of gastric diverticula employing the transthoracic approach is described.

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SURGICAL CORRECTION OF COARCTATION OF THE AORTA DURING PREGNANCY

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How should the combination of pregnancy and coarctation of the aorta be treated? On theoretical grounds alone, one would expect high mortality and morbidity rates when the increased load of pregnancy is superimposed upon the already overburdened cardiovascular systems of patients with coarctation. Recommendations for treatment have not been uniform but, by and large, have been concerned with interruption of the pregnancy or delivery by cesarean section to avoid the hazard of a long or hard labor.

Since the surgical correction of coarctation of the aorta was accomplished nearly 10 years ago[2, 4] it is rather surprising that we have found in the literature only one recommendation for operative attack on the coarctation rather than the pregnancy. This form of treatment was advocated by Miller and Falor[6] but in neither of the 2 cases reported by them in June 1952 was surgical correction of the coarctation attempted during the pregnancy.

In March of 1952 we were called upon to treat the first of the 3 patients with coarctation of the aorta associated with pregnancy which is the subject of this report. The indications for operative correction of an uncomplicated coarctation of the aorta had been established. It was our opinion that the presence of an early pregnancy was an added indication for, rather than contraindication to, immediate surgical repair of the coarctation. It was believed that this could be done without great risk to either the mother or fetus, and that, once it had been achieved, the patient should go on to uncomplicated, spontaneous delivery.

CASE REPORTS

Case 1. A 16 year old white female was admitted to the hospital on March 25, 1952 with a diagnosis of coarctation of the aorta. This diagnosis had been made at the U. S. Naval Hospital, Jacksonville, Florida, following study for a complaint of hypertension. Otherwise, the patient was asymptomatic.

On examination the blood pressure in the arms was found to be 160/78; that in the legs could not be recorded. No peripheral pulses were demonstrable in the lower extremities, but there were visible pulsations in the supraclavicular fossae. The heart was slightly enlarged to the left. No thrills could be elicited. There was a grade III systolic murmur heard over the entire precordium. This murmur was loudest in the second left intercostal space and was transmitted to the neck and back to a point just to the left of the midthoracic spine.

Left ventricular enlargement was confirmed by fluoroscopic examination. The electro-

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The opinions and conclusions expressed in this paper are those of the authors and are not to be construed as official or necessarily reflecting those of the Medical Department of the U. S. Navy, or of the Naval Service at large.

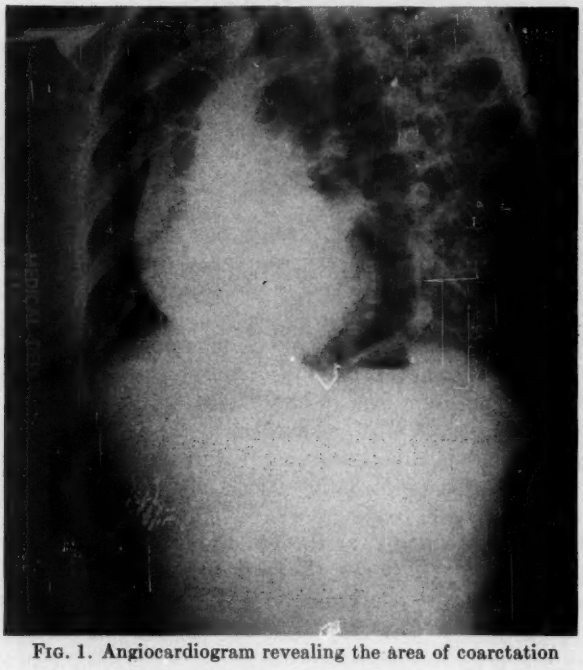

FIG. 1. Angiocardiogram revealing the area of coarctation

cardiogram showed left axis deviation. An angiocardiogram demonstrated the coarctation of the aorta just distal to the left subclavian artery (fig. 1). A pregnancy of six to eight weeks duration was suspected on the basis of the history and physical examination, and was confirmed by an Aschheim-Zondek test.

On April 15, 1952 the coarctation was treated by surgical excision and end to end anastomosis of the aorta. The postoperative course was uneventful and the patient was discharged from the hospital on April 29, 1952. At that time the blood pressure in the arms was 158/90 and in the legs was 140/90. Peripheral pulsations in the extremities were excellent.

On August 15, a follow-up communication stated that the patient was well and that pregnancy was progressing uneventfully. Blood pressures in the arms were 138/78 and 128/84; in the legs 148/94 and 134/92. The patient went on to spontaneous and uncomplicated delivery in October 1952.

Case 2. This 19 year old female was admitted to the hospital on July 18, 1952 with a complaint of *heart trouble*. This diagnosis had been made following a chest roentgenogram taken at an early prenatal examination one month previously. The history was noncontributory except for frequent dizzy spells in hot weather or following exercise.

The blood pressure in the arms was 160/100; in the legs no readings could be obtained on the sphygmomanometer. Femoral pulses were faint. Abnormal pulsations were elicited along the course of the ribs posteriorly and posterolaterally. No cardiac enlargement was demonstrable. There was a high pitched, grade I systolic murmur heard best along the left border of the sternum and in the left axilla.

The heart was normal in size to fluoroscopic examination. A roentgenogram of the chest showed notching of the ribs. The angiocardiograms were poor due to technical difficulties and did not contribute anything further to the clinical diagnosis of coarctation of the aorta. Abdominal and pelvic examinations revealed findings consistent with a three months pregnancy.

On July 30, 1952 the coarctation was resected and an end to end anastomosis of the aorta was made. During convalescence excellent pulsations were demonstrable in the lower extremities and blood pressures ranged from 154/100 to 160/100 in the arms, and from 168/68 to 150/68 in the legs. On the second postoperative day some vaginal spotting was noted but this cleared on progesterone therapy. She was discharged from the hospital on Aug. 13, 1952.

On Jan. 21, 1953 she delivered spontaneously a viable female child. During the first stage of labor blood pressure rose from 120/100 to 160/100, but after delivery it dropped to 128/86 and remained at this level during the postpartum period.

Case 3. A 24 year old white woman was admitted to the hospital on Jan. 5, 1953 with a diagnosis of coarctation of the aorta. The diagnosis had been made at the U. S. Army Hospital, Camp Pickett, when the patient was hospitalized there for bronchitis. Because of exertional dyspnea, palpitation, and a heart murmur the patient's activities as a child had been restricted. However, since the age of 16 she had lived a normal, active life without symptoms. At the time of admission she stated that she had not menstruated for three months and that she assumed she was pregnant.

Physical examination revealed a blood pressure in the right arm of 180/110 and in the left arm of 190/110. Blood pressures in the legs could not be recorded and pedal pulses were absent. The heart was enlarged to the left. There was a loud blowing systolic murmur at the base which was transmitted posteriorly and to the neck.

Fluoroscopy confirmed the cardiac enlargement and revealed an aortic outline consistent with the diagnosis of coarctation. A chest roentgenogram showed notching of the ribs.

Abdominal and pelvic examinations supported the patient's assumption that she was pregnant and a positive frog test was obtained. On January 8, a slightly bloody vaginal discharge was discovered and upon examination this was found to be coming from the cervix. The patient was placed at complete bed rest for 72 hours and given progesterone, 10 mg. twice daily. The bleeding ceased after a few hours.

On January 14, the coarctation was resected and continuity of the aorta was restored by end to end anastomosis. Postoperatively, pedal pulses were present and full. The blood pressure in the arms was 140/90 and in the legs 155/90. Convalescence was complicated by a penicillin reaction and one very short episode of vaginal bleeding but both of these had cleared at the time of discharge on February 9.

On July 16, the patient delivered spontaneously and made an uneventful postpartum recovery. On Nov. 2, 1953 blood pressures in the arms were reported as 125/70 and 145/75; in the legs as 150/90 and 145/85.

DISCUSSION

Although coarctation of the aorta was described by Paris[7] in 1791, there has been relatively little written about this condition in association with pregnancy. In 1949 Benham[1] reviewed the literature and found only 56 such cases. Since that time Greig and Parker[3] have reported 1 case and Miller and Falor[6] 2 cases. These cases added to the case of von Lindeboom[5] which was published in 1947, but not included in Benham's review, bring to 60 the total number of cases available for study.

Casual perusal of this literature would lead one to believe that the maternal mortality rate is extremely high and that the risk of carrying the fetus to term is very great when pregnancy is associated with coarctation. However, a careful review of the cases reported does not support this conclusion in that they have not been documented sufficiently clearly for a group of patients large enough to yield significant data on the over-all risk.

Obviously a number of factors are involved. Although there may be some theoretical basis for arguing that the presence of coarctation should predispose

to the development of toxemia in pregnancy, the complications which have been observed in association of the two conditions are essentially those of coarctation alone. These include rupture of the distal or proximal aortic segment or one of the major collateral channels, cardiac failure, and cerebral vascular accidents. Probably the threat of major complications of coarctation in pregnancy has been somewhat exaggerated. Several patients have been seen who have had multiple pregnancies with normal deliveries prior to diagnosis or treatment of the coarctation. On the other hand it is quite certain that the patients who have developed dilatation and thinning of the distal aortic segment or the collateral intercostal vessels are subjected to a severe hazard in the later months of pregnancy and during labor.

Since the potential dangers of coarctation per se are constantly present, and since it is impossible to determine for any given case just how much the superimposed pregnancy increases the hazard, we believe that it is an extremely wise policy to intervene electively in all cases of coarctation in which pregnancy occurs. The end of the first trimester would appear to be the optimum time for surgical correction of the coarctation because of the stability of the pregnancy at this stage. It has been with these considerations in mind that we elected to proceed in the cases reported here. The successful outcome in all 3 patients is highly gratifying and at the present time we recommend this as the treatment of choice.

SUMMARY

A study has been made of the literature pertaining to the association of coarctation of the aorta and pregnancy.

Measures to treat this combination in the past have included interruption of the pregnancy or delivery by cesarean section to avoid the hazard of a long or hard labor.

It is believed that surgical correction of the coarctation early in the pregnancy is a more logical form of treatment and that it can be accomplished without great risk to either the mother or fetus.

Three cases have been reported to support the above contention. In each instance the coarctation of the aorta has been successfully resected at about the third month of pregnancy and each patient has gone on to an uncomplicated spontaneous delivery of a living child.

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HEMORRHAGIC TELANGIECTASIS OF THE GASTROINTESTINAL TRACT; AN OBSCURE SOURCE OF GASTROINTESTINAL BLEEDING

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Massive bleeding from telangiectasis of the gastrointestinal tract, although uncommon, may present a difficult problem in diagnosis and treatment. Usually gastrointestinal telangiectasia is a manifestation of a more generalized disease recognized as hereditary hemorrhagic telangiectasis. More widespread recognition of this disorder is desirable since unsuccessful exploratory operations for gastrointestinal bleeding are sometimes the result of failure to appreciate the telangiectatic origin of the bleeding.

HISTORICAL BACKGROUND AND CLINICAL COURSE

Rendu,[41] Osler,[35] and Weber[58] first called attention to this entity about the turn of the century. Since that time over 1000 cases of the disease have been reported in the literature.[28] The classical diagnostic features are: (1) presence of telangiectasis, (2) tendency for the lesions to bleed, and (3) familial history of bleeding. About 20 per cent of the recorded cases have lacked a family history.[54] This familial history generally is not considered necessary to make a diagnosis of the disease.[8] Transmission is by Mendelian dominant gene which is not sex bound, and has been followed through six generations in several instances.[7, 54, 56] Fitz-Hugh[9] regarded nonhereditary cases as atavistic instances of the disease.

Typically, nosebleeds begin in childhood, or shortly after puberty, and plague the afflicted person intermittently for the rest of his life. However, the telangiectasis may be found in one or more visceral organs and, therefore, gastrointestinal bleeding, hematuria, or hemoptysis may be the outstanding symptom. Visible lesions are first noted in the second decade with maximum development of the lesions and symptoms in the fourth decade. The telangiectasis may be punctate, spider-like, or nodular in shape and bright red in color. They are most commonly found on the mucosa of the nasal septum, tongue, lips and mouth. Cutaneous lesions are most common on the face, scalp, chest and fingertips. The telangiectasis range from 2.5 to 5 mm. in diameter and may undergo spontaneous regression followed by development of new lesions in the nearby area. The telangiectasis fades on pressure. Purpuric spots can be differentiated by this maneuver. Hepatosplenomegaly commonly is present in the advanced

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stages of the disease, and in those people with the more severe form of the disease. The mortality rate due to exsanguinating hemorrhage is about 4 per cent.[9] The morbidity rate of the disease is much greater. Due to chronic blood loss many patients become debilitated and die of intercurrent infection.

PATHOLOGIC PHYSIOLOGY

Hereditary hemorrhagic telangiectasis is most widely thought to be due to a congenital developmental dysplasia of the vascular system. In certain capillary beds there is a deviation from normal of capillary formation resulting in anatomically defective vessels.[52] There is an absence of the muscular coat of these anomalous vessels causing ballooning or dilatation of the walls. Histologically such a defective vessel appears as a dilated thin walled channel lined by a single layer of endothelium (figs. 1 and 2). Muscle and elastic fibers are notably absent from the vessel wall. Prolonged bleeding is thus explained by inability of the telangiectatic vessel to contract due to absence of these fibers. The bleeding time of the systemic capillaries is normal.

GASTROINTESTINAL MANIFESTATIONS OF HEREDITARY HEMORRHAGIC TELANGIECTASIS

The incidence of hemorrhagic gastrointestinal telangiectasis has not been stated previously. In an attempt to determine the approximate incidence, 100 case reports of this disease found in the literature were studied. Since one could accumulate an incorrect sampling by attempting to collect all case reports which mentioned gastrointestinal bleeding in the title of the report, only case histories simply listed as hereditary hemorrhagic telangiectasis were reviewed. A case history was considered acceptable for tabulation if it was apparent from the report that the patient gave a history of melena and/or hematemesis not attributed to other gastrointestinal pathology. This patient was tabulated as bleeding presumably from gastrointestinal telangiectasis. Of the 100 cases analyzed, 17 per cent of the patients gave a history of having gross gastrointestinal hemorrhage for which no other cause was found (table 1). There were at least 3 patients in this series with occult bleeding that caused severe secondary anemia. These figures indicate a greater incidence of bleeding from gastrointestinal telangiectasis than has been estimated by some investigators.[13, 28]

DIAGNOSIS

The telangiectasis may diffusely involve the mucosa of the gastrointestinal tract or be localized to a particular segment. In Osler's[35] original report, the autopsy findings of telangiectasis limited to the gastric mucosa was mentioned in one instance. Boston[5] made a similar observation. It is possible that, although the telangiectasia is diffuse, only certain lesions may bleed i.e., those most heavily traumatized by the digestive and elimination processes, (stomach and rectum). In a patient with the classical features of hereditary hemorrhagic telangiectasis who has gastrointestinal bleeding, a presumptive diagnosis of hemorrhagic gastrointestinal telangiectasia logically may be made by excluding other causes of

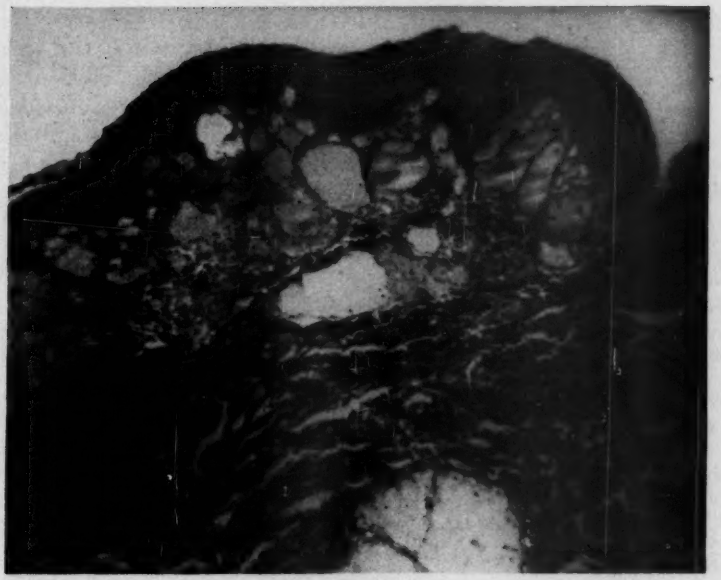

FIG. 1. Low power photomicrograph showing elastic tissue stain of telangiectasis. Removed from anterior chest wall of patient described in case history.

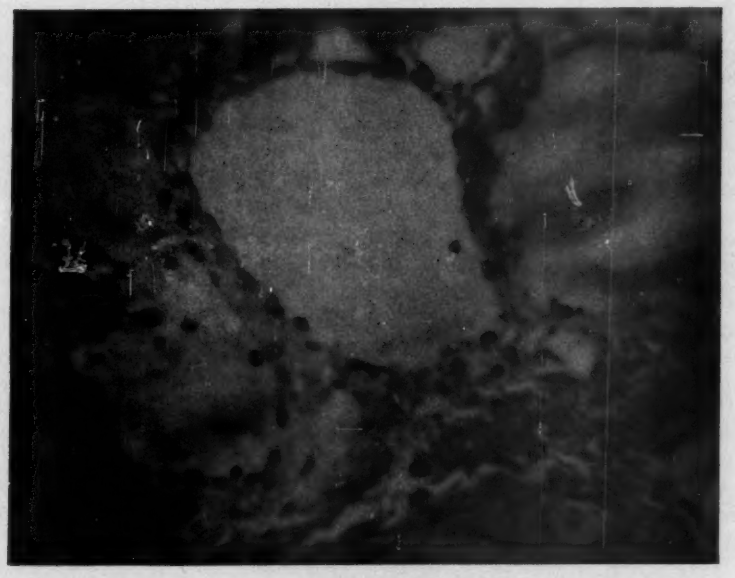

FIG. 2. High power photomicrograph with elastic tissue stain of same lesion. No elastic fibers are seen in the dilated vessel wall.

TABLE I

Incidence of hemorrhagic gastrointestinal telangiectasis

Author	Number of Cases Reported	Hemorrhage Presumably From Gastrointestinal Telangiectasia
Osler[35, 36]	4	0
Fitz-Hugh[9, 10]	5	2
Hurst et al[23]	8	2
Meikle[32]	1	1
Schuster[48]	1	0
Kelly[24]	2	0
Hawthorne[20]	1	0
Richardson[43]	2	1
Hanes[19]	7	0
Schwartz, V. J.[50]	1	0
Steiner[53]	5	0
Koch et al[27]	5	1
Schwartz, S. O. et al[26, 49]	2	0
Rundles[46]	1	1
Armentrout and Underwood[1]	2	1
Glass[15]	5	2
Gambocorta[11]	3	0
Goldman et al[17]	2	0
Miles[33]	1	0
Petch[38]	1	0
McEwan[30]	1	0
Wolfson[59]	1	1
Cappon[6]	1	0
Kennedy[25]	1	0
Peluse[37]	1	1
Stock[54]	7	1
Gitlow and Frosch[14]	4	2
Teahan[56]	1	0
Barrock[3]	2	0
Larrabee and Littman[29]	5	0
Voyles and Ritchey[57]	2	0
Saksela et al[47]	13	1
O'Kane[34]	1	0
Storey and Akamatsu[55]	1	0
Total	100	17

gastrointestinal bleeding. A positive diagnosis can be made only if the lesions are within range of the gastroscope or the sigmoidoscope or demonstrated at laparotomy. There are excellent descriptions in the literature of both gastric[17, 23, 42, 45, 46] and rectosigmoidal telangiectasis.[23, 57] Usually, these lesions appear very similar to those seen in the buccal mucosa. It must be remembered that gastrointestinal telangiectasis may occur without any other stigmas of the disease.[40]

TREATMENT

Hereditary hemorrhagic telangiectasia is not a disease of the blood but of the blood vessels. Successful treatment of bleeding lesions in any part of the body

must necessarily include removal or eradication of the telangiectasis. Bleeding points in the nose and mouth have been treated variously by direct electrocauterization or chemical cauterization, irradiation and local excision with little success due to the formation of new telangiectasis in the adjacent mucosa. Hemorrhagic telangiectasis of the more inaccessible gastrointestinal tract presents a more difficult problem. Treatment of hemorrhage from these lesions can be divided into medical and surgical measures. The great majority of people with hemorrhage from the gastrointestinal tract simply have been given supportive therapy including the administration of multiple blood transfusions. Such treatment is of great benefit to the low grade bleeder, but is insufficient to save the persons with exsanguinating hemorrhage. Kushlan,[28] in 1946, reported a decrease in the tendency toward recurrent bleeding following the administration of rutin. However, Glass,[15] following a carefully controlled group of 6 patients, noticed no beneficial result from rutin administration. Estrogens also have been administered with inconclusive results.[27]

The surgical approach to this disease is varied. Only a few pertinent results are found in the literature. Massive hemorrhage from gastric telangiectasis has been treated surgically in 3 patients; each time the surgeon employed a different method. Boston[5] did a devascularization procedure, ligating the vascular supply of the telangiectatic area in the gastric mucosa. The patient had no further bleeding in a five year follow-up. In 1944, Macklin[31] referred to a case in which a large, thin-walled, telangiectatic vessel in the gastric mucosa, which had ruptured, was locally excised. The patient recovered and his gastric hemorrhages stopped. In 1953, Shepherd[51] cauterized bleeding telangiectasis of the remnant of gastric mucosa. The patient previously had a subtotal gastric resection for duodenal ulcer. He died of massive hemorrhage on the fourth postoperative day. Shepherd also reported a case in which he found widespread telangiectasis of the serosal surface of the small bowel while doing a palliative gastric resection for gastric carcinoma. The patient died on the second postoperative day. The cause of death was not known. Ferguson[4] did a right hemicolectomy for multiple small bleeding points in the cecum and ascending colon. The pathologic diagnosis was submucosal telangiectasis. The patient had no further bleeding in a 19 month follow-up. Mention should be made of three instances in which exploratory operation was done for unexplained gastrointestinal bleeding before the correct diagnosis of hereditary hemorrhagic telangiectasis could be made.[18, 37, 51] In each patient, the operation was done during a nonbleeding period and the cause of the hemorrhage was not found. It is probable that the bleeding in each patient was from gastrointestinal telangiectasis. Small lesions can be discovered best if a laparotomy is done during a bleeding period. At that time, close examination of the mucosa at the upper end of the column of blood in the gastrointestinal tract is of great aid in detecting the small bleeding points.[4, 21]

CASE REPORT

A 49-year old white man entered the hospital on Jan. 18, 1954 complaining of vomiting blood and passing dark stools. He was admitted to the surgical service

because of the history of recurrent bleeding and suspected duodenal ulcer. In May of 1950, he passed tarry stools for a period of eight days. He had a similar episode in May of 1951. In August of 1953 he vomited blood and passed tarry stools for two days. He was told by doctors he should have a portion of his stomach removed. Because of further hematemesis on Jan. 4 and 18, 1954, he came to this hospital. He had received, in the past, a total of 6,500 cc. of blood. A cause for his bleeding had never been discovered.

At the time of admission the patient was pale and apprehensive. The blood pressure was 156/80 and the pulse was 88. Except for the signs of marked blood loss and hepatosplenomegaly, physical examination revealed no abnormalities. The red blood count was 3,100,000 per cu. mm. and the hemoglobin was 7.6 Gm. per 100 cc. (46 per cent). He passed a tarry stool on the second hospital day. He then had no further bleeding. He received a total of 4,000 cc. of blood to replace the blood loss. Careful roentgenologic examinations of the gastrointestinal tract, including special studies of the lower esophagus, duodenum and small bowel, were negative. Liver tests showed normal liver function. Liver puncture biopsy showed normal liver tissue. All laboratory studies, including platelet count, bleeding time, and coagulation time, were normal. On review of previous records it was found that similar examinations had been done repeatedly in the past with negative results.

On the third hospital day the patient had a spontaneous nosebleed. Detailed questioning revealed a familial history of nosebleeds which previously had not been elicited (fig. 3). He had his first nosebleed at the age of 14 and until the age of 22 he had at least one nosebleed daily. Between the ages of 22 and 39 he had one to two nosebleeds weekly and after that time had about one nosebleed per month. The patient's father and daughter had nosebleeds all of their lives. This familial history suggested the diagnosis of hereditary hemorrhagic telangiectasia and this was confirmed by finding the typical lesions on the skin and mu-

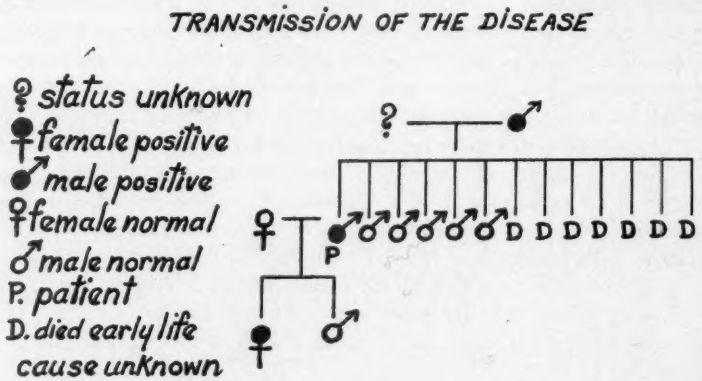

FIG. 3. Family tree showing transmission of hereditary telangiectasis through three generations.

cous membranes of the patient which had been missed on previous physical examination. The lesions were small, bright red and punctate and were more numerous on the tongue and lips. Scattered spider-like lesions were seen on the skin of the chest wall, scalp, and nasal septum. The patient's daughter was also examined. She had many lesions on the tongue, lips, nasal septum and buccal mucosa but no cutaneous involvement.

In view of the patient's negative gastrointestinal studies, a presumptive diagnosis of hemorrhagic telangiectasis of the upper gastrointestinal tract was made. Gastroscopy showed a single, small, punctate telangiectasis in the lower portion of the stomach on the anterior wall. Also, close by, a slightly elevated, bluish lesion was seen appearing much like an isolated varix. Another varix-like lesion was seen on the lesser curvature. No gastric ulcerations or new growths were seen.

DISCUSSION

In this particular case, duodenal ulcer and esophageal varices were excluded as the source of bleeding by roentgenogram and laboratory studies. The normal blood studies ruled out the various hemorrhagic diseases. The fortuitous nosebleed led to the diagnosis. Surgical intervention was not indicated since the patient stopped bleeding shortly after admission.

The nature of the disease was explained to the patient and he was instructed to return to the hospital in the event of further gastrointestinal bleeding. If hemorrhage in the future is massive and persists after initial restoration of blood volume, immediate surgical exploration is contemplated. The surgical procedure to be employed will depend upon the number and distribution of the bleeding telangiectases.

Mention should be made of the frequent association of hereditary hemorrhagic telangiectasis with other vascular anomalies. Occurrence of multiple congenital defects, particularly in the same body system, is common. The occurrence of congenital varices, congenital aneurysm, (usually cirsoid), arteriovenous fistula or other vascular anomalies may be regarded in the patient with hereditary hemorrhagic telangiectasis as a further expression of the inherited tendency toward vascular dysplasia. Arteriovenous fistula of the lung now is considered in many cases to be a visceral manifestation of hereditary hemorrhagic telangiectasis.[2, 16] Associated aneurysm of the splenic artery[43] and also vascular anomalies of the portal vein have been noted. Interestingly enough, the associated anomaly of the portal vein produced an extrahepatic type of portal obstruction in a father and son with hemorrhagic telangiectasis.[44] Associated vascular anomalies of the gastrointestinal tract have not been mentioned in the literature. The vascular lesions described by Boston[5] and by Macklin,[31] and the varix-like lesions seen on gastroscopy in the case reported here, most likely represent associated anomalies of the larger blood vessels. Rathmell[39] described the case of a woman who died of a fatal hemorrhage from a congenital aneurysm of the jejunum. It was noted that her father suffered from recurrent attacks of epi-

staxis of undetermined cause. A diagnosis of hemorrhagic telangiectasis was not mentioned, but is suggested by the presence of vascular defects in two successive generations.

The occurrence of hepatosplenomegaly in this disease has not been satisfactorily explained. Liver function studies usually are normal. Telangiectases of these viscera and hemopoietic response to anemia have been offered as possible explanations.

SUMMARY

A review of 100 case histories of patients with hereditary hemorrhagic telangiectasis revealed that 17 per cent gave a history of massive hemorrhage presumably from telangiectasis of the gastrointestinal tract.

Hemorrhage from telangiectasis of the gastrointestinal tract may be massive and require surgical intervention. Exploration should be done during a bleeding period.

Hepatosplenomegaly is seen in the more severe form of the disease.

Hereditary hemorrhagic telangiectasis often is associated with other vascular anomalies.

This disease should be considered in every case of obscure gastrointestinal bleeding.

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MASSIVE INTRAPERITONEAL HEMORRHAGE FROM ACUTE CHOLECYSTITIS WITH PERFORATION*

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The subject of perforation of the gallbladder is of great practical significance. "On its frequency and dangers depends much of the decision as to when to operate in acute cholecystitis." This statement by Cowley and Harkins[2] in 1943 succinctly poses the problem presented by acute cholecystitis. More recently, Schmitz, Schlosser, and Harkins[9] (1953) reviewed the experience at King County Hospital, and concluded that perforation occurs in an appreciable number of patients in the older age group with a resultant high mortality rate. They recommended more frequent interval cholecystectomy, and more frequent early operations for acute cholecystitis.

We wish to report one of the rarer, but very serious, complications of acute cholecystitis—namely, perforation with massive intraperitoneal hemorrhage. While we have done but 48 operations for biliary tract disease from 1951 to 1953, 11 of these patients had acute cholecystitis, and of these 11, 4 had perforated. Two of the perforations resulted in local abscesses, 1 resulted in a subdiaphragmatic abscess, and the fourth case, to be cited here, was associated with massive intraperitoneal hemorrhage. In this very small series of patients with acute cholecystitis, 7 were treated by cholecystostomy with no deaths, while 1 of the 4 subjected to cholecystectomy developed fatal pulmonary edema during the course of the operation. Of the 4 perforations, 3 were treated by cholecystostomy, and 1 required only subphrenic space drainage.

CASE HISTORY

H. T. A., a 60 year old white man—a chronic alcoholic—was admitted to the Seattle Veterans Administration Hospital on Oct. 16, 1952. He had been well until six days before entry when he noted a rather sudden onset of diffuse epigastric pain. This pain soon became more severe in the right upper quadrant, where it persisted as an unrelenting steady ache until admission. He had vomited occasionally, but for the most part had tolerated a soft diet.

His temperature was 102.4 F., pulse 136, and blood pressure 152/90. The abdomen was distended and tympanitic. Exquisite tenderness with positive rebound phenomenon was present over the entire epigastrium, but was most marked in the right upper quadrant. No masses were palpated. Peristalsis was moderately high pitched and sluggish.

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The urinalysis was normal; the hematocrit was 30; and the white cell count was 11,000 per cu. mm. with 92 per cent neutrophils. A chest film revealed streaks of segmental atalectases, and a flat abdominal film showed only generalized ileus. The serum amylase was 4 (normal in our laboratory 100–150).

The patient was treated conservatively for 36 hours with intestinal suction, antibiotics, blood and fluids. During this time he passed one liquid dark maroon stool which was strongly guaiac positive. Sigmoidoscopy was normal. At the end of this period the general condition was improved, although his temperature still was 102 F, and a vague mass had developed in the right upper quadrant which still was exquisitely tender.

On October 19 he was operated upon. After the induction of gas-oxygen-ether anesthesia with endotracheal intubation, an ill-defined spheroid mass, approximately 15 cm. in greatest dimension definitely was apparent beneath the right subcostal border. A transverse incision was made directly over this mass lateral to the right rectus muscle. On opening the peritoneum 1500 cc.—measured—of dark red liquid and clotted blood was removed from the right subhepatic space. The incision then was extended medially with transection of the right rectus muscle and linea alba to afford exposure for determination of the source of hemorrhage. Exploration revealed a small, contracted, firm, nodular liver (from the free edge of which tissue for biopsy was taken), dilated omental and gastroepiploic veins, an enlarged spleen, and a recently organizing hematoma about 14 cm. in diameter surrounding the gallbladder. On entering this hematoma to inspect the gallbladder, brisk bleeding was encountered which could be controlled only by packing. The source of bleeding was an acutely inflamed, friable, edematous gallbladder which had ruptured along its lateral hepatic attachment from its fundus to its ampulla. Cholecystectomy was considered as a means of arresting the hemorrhage, but accurate dissection could not be done safely because of extensive inflammation of the hepatoduodenal ligament and old and recent vascular adhesions fixing the distal stomach and duodenum to the gallbladder. Accordingly, the fundus of the gallbladder was resected. Hemorrhage continued, however. It seemed to arise from the lumen of the ampulla and cystic duct. No bile was seen. Stones could not be felt. Attempts to stop the bleeding by clamping and suture ligature were unsuccessful because of the extreme friability of the tissue and lack of any definite bleeding point. Hemorrhage was controlled only by insertion of a pack extending through the lateral border of the wound. The wound was closed in layers with interrupted wire sutures. Two and one-half liters of blood were required during and immediately after the operation to maintain adequate blood volume.

Microscopic sections of the gallbladder showed marked thickening by edema and proliferation of fibrous tissue. An acute, fibrinous, inflammatory exudate covered both mucosal and serosal surfaces. There was infiltration of the entire wall by inflammatory cells consisting principally of neutrophils. Perivascular accumulations of leukocytes were seen around many subserosal vessels; one small artery near the surface contained a recent fibrin thrombus. The liver biopsy showed lobulation by fibrous tissue, connecting the portal triads, which contained many chronic inflammatory cells. Pathologic diagnoses were chronic cholecystitis with superimposed acute inflammation, and portal cirrhosis.

The immediate postoperative course was complicated by a moderate hepatocellular jaundice which subsided after 10 days. The pack was withdrawn in stages without further bleeding. Despite cautious parenteral therapy and attempts to provide a high carbohydrate and protein intake, peripheral edema and ascites, with a low serum sodium and albumin, appeared on the fourth postoperative day and persisted for a month. Barium swallow and esophagoscopy during this period showed esophageal varices.

The patient was discharged on December 6 (eight weeks postoperative), but was rehospitalized from Jan. 13, 1953 to Feb. 12, 1953 for another episode of hepatocellular jaundice. Repeat esophagoscopy on April 30, 1953 (six and one-half months postoperative) failed to demonstrate the varices, and it is postulated that the patient's inflammatory process and surgery may have allowed for the development of extensive collateral circulation. On May 5, 1953 an incisional hernia was successfully repaired, at which time many

vascular adhesions were noted. The patient enjoyed good health until Jan. 6, 1954 when he was confined on the psychiatric service for alcoholism and depression.

DISCUSSION

Massive hemorrhage from a perforated gallbladder is rare. We have been able to find only 10 other reports since Leared's original case report in 1858.[6] These cases are summarized in table I.

Analysis of these 12 cases (11 in literature plus our own) reveals that 7 of the 12 died (58.3 per cent mortality). However, only 1 of the 4 patients since Mailer's[7] report in 1939 died; this latter group of 4 was treated from 1947 to the present, and had the advantages of multiple blood transfusions and antibiotics. Three of the 12 patients were treated without operation; all of these 3 died. Nine patients were operated upon, and 5 of these recovered. Control of hemorrhage at the time of operation constituted a definite problem in 5 of the 9 surgically treated patients, and was accomplished in all but 1 patient (including the authors') by a pack inserted under pressure and left *in situ*. This crude but time-honored method of controlling hemorrhage still has merit. Valuable time and blood were wasted during the authors' operation in attempts to stop bleeding by clamping, suture ligatures, and partial cholecystectomy.

Although in this case report the diagnosis of acute cholecystitis was considered preoperatively along with other entities, the correct diagnosis of acute cholecystitis *with perforation and massive hemorrhage* was not made until operation. In retrospect, the history, the anemia, the passage of a bloody stool, and the finding of a *large* right upper quadrant mass all indicated the unexpected operative findings.

Mailer's discussion in 1939 is one of the clearest in elucidating the mechanism of perforation and associated hemorrhage. In a study of 30 acutely inflamed gallbladders he reported that, "The main findings were venous engorgement, edema, and small hemorrhages in the milder cases, and gross hemorrhages and even hemorrhagic infarction in the most severe. . . . In only half of the cases showing venous and lymphatic engorgement and hemorrhage was there evidence of superadded bacterial infection." He concluded that, ". . . the main factor in the production of the majority of acute gallbladder lesions is primarily vascular, dependent upon the impaction of a gallstone at the neck of the gallbladder." The added effect of portal hypertension as a factor in the pathogenesis of the case reported herein is questionable. From the above description, one may postulate that massive hemorrhage is more likely to result from an acutely inflamed gallbladder with previously engorged vessels than from one without the presence of portal hypertension.

Concerning the treatment Mailer stated, ". . . If the facts as stated receive confirmation and acceptance, there is prima facie strengthening of the case for early operation upon the acute gallbladder, at a time when there is a fair chance of operating in a noninfected field. In cases seen at the end of 48 hours, and which are subsiding, delay for two or three weeks seems advisable before operation is considered, as this will give time for the gallbladder to sterilize itself. The chief

TABLE I

Resume of twelve case histories of acute cholecystitis with perforation and massive intraperitoneal hemorrhage

Author & Date	Sex & Age	Operative Findings & Procedure	Outcome	Autopsy Findings
Leared[6] 1858	22 Male	None	Death on 13th day of illness	Gangrenous cholecystitis with perforation. Two liters of blood in peritoneal cavity. Stone in common duct. No particular vessel identified as source of bleeding.
Schnyder[10] 1915	72 Male	None	Death on 2nd day of illness	Acutely ulcerated gallbladder with perforation and erosion of vessels. Two liters of liquid and clotted blood in peritoneal cavity.
Gjellerup[4] 1921	72 Female	Perforation of gallbladder with one liter of blood in peritoneal cavity	Recovery	
Waters[11] 1926	63 Female	Perforation at the ampulla and tear of the cystic artery with free blood and stones in the peritoneal cavity	Recovery	
Wyse[12] 1934	79 Male	None	Death on 6th day of illness	Acute cholecystitis with gangrene and perforation. Cholelithiasis and stones free in peritoneum. There were 700 cc. of blood in peritoneal cavity and large hematoma around gallbladder.
Bartlett[1] & Bartlett 1936	65 Female	Perforation with stones and two liters of blood in the peritoneal cavity.	Death on 3d postoperative day	No further hemorrhage. Chronic cholecystitis with edema. Death from "pneumonia".
Sanders[8] 1937	45 Male	Gangrenous cholecystitis with perforation and many large clots of blood.	Death on 3d postoperative day from old pneumonitis and nephritis	Not reported.
Mailer[7] 1939	65 Male	Acute cholecystitis with perforation and active bleeding of 1.5 to 2 liters of blood in peritoneal cavity	Death 3 hours postoperative in shock	Cultures of gallbladder sterile. Acute cholecystitis and gangrene adjacent to the perforation

Hardy & Spelman 1947	52 Female	Acute perforation with 1300 cc of liquid and clotted blood	Recovery	
Fitts, DeMuth, Ravdin[3] 1951	66 Male	Gangrenous cholecystitis with 1500 cc of blood in the peritoneal cavity	Death in 36 hours post-operative from shock and probable myocardial infarction	Permission for autopsy refused
Idem. 1951	71 Female	Acute cholecystitis with a large hematoma surrounding the gallbladder	Recovery	
Authors' 1953	60 Male	Described in text	Recovery	
Total, 12 cases	Average age 61. Nine of twelve 60 or over	Nine patients operated upon. Four partial cholecystectomies. Cholecystostomy in 11. Five necessitated control of active hemorrhage accomplished in 4 instances by pressure with a pack left *in situ*	Seven deaths. Five recoveries. Mortality rate 58.3%	

difficulty arises in old people in whom the surgeon's natural instinct to delay must be tempered with the knowledge that in patients in the sixth and seventh decades of life severe vascular lesions going on to gangrene and perforation are particularly liable to occur."

This point of view was re-emphasized by Fitts and associates[3] (1951), and is supported by the authors. Nine of the 12 patients were 60 years of age or over. From the fact that a larger percentage of the population is reaching more advanced years, it may be expected that more individuals treated without operation will develop perforation of the gallbladder. In this group there will be a certain proportion with the additional hazard of hemorrhage.

One reads and hears many medical and surgical discussions concerning the advantages of the operative and nonoperative methods of managing acute cholecystitis. We believe that, while early cholecystectomy represents the ideal definitive treatment, it is unfortunate that many proponents of *early operation* do not give enough emphasis to the benefits of cholecystostomy. When one operates too late in the course of acute cholecystitis for safe cholecystectomy, cholecystostomy still represents a life-saving means of averting gangrene and perforation. It behooves us, then, to pursue a course of aggressive surgical treatment in preference to a *wait and see* policy, especially in the elderly patient.

SUMMARY

The literature pertaining to hemorrhage as a complication of acute gallbladder disease is reviewed and an additional case is reported. The hazard of this complication with its high mortality rate is noted.

Early cholecystectomy or cholecystostomy is recommended to prevent the complications of acute cholecystitis, especially in the elderly patient.

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MANAGEMENT OF INTRAORAL CANCER

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By intraoral cancer we mean cancers occurring from the soft palate and anterior pillars of the fauces forward to the inner side of the lips.

For purpose of discussion, intraoral cancer may be grouped according to location in: Tongue and floor of mouth; buccal mucosa and lower gingiva; and palate and upper gingiva. The lesions in each group have similar development, treatment and prognosis. Statistically, the death rate from intraoral cancer is about 1.4 per cent per hundred thousand population.

Many etiologic factors—some still controversial and unproved—may contribute to the development of carcinoma of the oral cavity. Perhaps the most common one is trauma. There is some as yet unknown biologic factor which prepares the soil—so to speak—because trauma occurs in many patients who never develop cancer. Ragged teeth and poor dental hygiene often are associated with cancer of the buccal mucous membrane. Malfitting, loose or sharp-edged dentures frequently overlie a malignant ulcer. Tobacco certainly is an irritant, although its exact role still has not been established. Last, the relationship of leukoplakia to cancer in the oral cavity is readily admitted. The authors at present are engaged in a long-termed project to discover the exact relationship between leukoplakia and cancer of the mouth.

The three most prominent lesions of the oral cavity to be distinguished from cancer are the chronic ulcer found in tertiary syphilis and tuberculosis, and ulcerated thickened leukoplakia. Other lesions, such as psoriasis, lichen planus, granulomas, and the acute ulcers of trauma and Vincent's angina, secondary syphilis, and aphthomatous ulcers may require differentiation from cancer. Since the differential diagnosis has been adequately and extensively described in the numerous texts and papers on the subject, we will not elaborate. Suffice it to say, adequate biopsy and subsequent accurate pathologic study is the only real diagnostic procedure that determines the presence or absence of cancer.

In the majority of cases, the clinical diagnosis of malignant disease in the oral cavity is not difficult to the trained eye and finger. Despite intensive educational propaganda, both lay and medical, there are far too many patients referred with extensive disease.

The most common malignant tumor of the oral cavity is squamous cell carcinoma, which may be papillary, sessile, ulcerative or infiltrative in character. Adenocarcinoma may arise from embryonic mucous gland epithelium or mucosal salivary glands, and usually involves adjacent bone early. Carcinoma, or sarcoma, may arise from aberrant mixed salivary tissue tumors. Sarcoma per se is an extremely rare lesion in the mouth. The authors have had 3 cases of

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malignant melanoma primary in the oral cavity. Systemic diseases such as lymphosarcoma and leukemia occasionally are first noted in the mouth.

Except for a selected group of early lesions, the best chance of cure lies in the field of radical surgery, provided it offers a reasonable degree of comfort, function and rehabilitation. For the patient is little better off despite the eradication of his cancer if the results of radical surgery turn him into a social and economic outcast. The authors, in so far as possible, carry out a well planned plastic reconstruction at the time of the radical extirpation of the tumor. Thus, radical surgery should aim at two goals: (1) removal of the primary tumor, along with the primary pathways of lymphatic spread, and (2) the cosmetic and functional rehabilitation of the patient. Radical extirpation of the cancer should be without consideration of important but not essential anatomic structures (internal carotid artery). For uncontrolled cancer of the oral cavity unequivocally is a fatal disease.

Although many of the technical procedures employed today first were conceived by Kocher, Butlin and Crile, Sr.[12, 1, 3] and others it is only since the advent of antibiotics, the advancement is anesthesiology, the unlimited supplies of whole blood, and the facilities for sustaining parenteral feedings for long periods of time, that such procedures have become feasible. During the past decade operative mortality and morbidity rates have been reduced to such an extent to make radical surgery for mouth cancer practicable.

CARCINOMA OF TONGUE AND FLOOR OF MOUTH

Embryologically, the tongue and floor of mouth arise from the first and second branchial arches. The clinical behavior of cancer in each location is similar except that floor of mouth cancer frequently involves the mandicular periosteum and metastasizes earlier to the upper neck.

A few small cancers of the tongue are treated satisfactorily by irradiation therapy, or wide local excision, especially with electrosurgery, and an occasional small cancer of the floor of the mouth can be eradicated with irradiation therapy. Usually, tumors so treated are 1 to 2.5 cm. in greatest diameter and located in the anterior part of the mouth and anterior third of the tongue. Intraoral x-ray cones have a limited place in the treatment of an occasional small cancer on the anterior one-third of the tongue. Great care must be exercised in focusing the cone and maintaining the application accurately. Cancer of the floor of the mouth cannot be treated adequately with an intraoral x-ray cone. Radon seeds and radium element needles are of great value in eradicating carefully selected small cancers of the anterior third of the tongue and an occasional cancer of the floor of the mouth.

The patient should wear, for 10 or 12 days, a lower denture having a built-in lead shield which covers the area containing the seeds and protects, to some extent, the hard palate and upper gingiva and, if possible, the mandible. When radium needles are used, the lead shield is worn only as long as the needles are in place. The dose should be calculated according to one of the accepted standard dosage tables (Paterson and associates and Quimby and associates).[5] We

have had no experience with the use of radium moulds applied externally for protracted treatments as practiced by English radiologists.

An ever-present danger following implantation of radon seeds or radium element needles is the complication of radio-osteo-necrosis of the mandible which may appear from two months to two or three years, or occasionally longer, following treatment. To lessen the possibility of this undesirable and painful process, all teeth near the area of disease should be extracted prior to treatment.

Elderly or debilitated patients form another group in whom irradiation is at times preferable to surgery for cancers of the tongue and floor of mouth, both small and extensive.

In the majority of patients with cancer of the tongue and floor of mouth, we now employ immediate radical surgery for resectable lesions. Occasionally, preoperative irradiation therapy is given for those lesions that are too large for a successful cancer operation. The operation of choice is hemiglossectomy and neck dissection en bloc, either by the composite, or the pull-through procedure. The possible exceptions are: (1) small local cancers on the tip of the tongue, particularly papillary in type. These are slow to metastasize, noninfiltrative and yield to wide, local excision. (2) extremely poor operative risks, in the aged and debilitated. Even here the oft-quoted phrase of Jules Able, "There are no medical contraindications to cancer surgery", must be borne in mind. (Quoted from Martin).[7] (3) patients who refuse surgery.

Our reasons for no longer adhering to the combination of preoperative irradiation and surgery or irradiation alone for the treatment of the local growth are: (1) some 25 per cent of excised local growths in the mouth following adequate irradiation still retain viable cancer cells;[12] (2) many patients will refuse the much needed neck dissection following the successful alleviation of the primary growth and its symptoms by irradiation; (3) radiation therapy given in sufficient quantities to eradicate cancer in the mouth causes permanent damage in the surrounding tissues, resulting in a high incidence of secondary necrosis; (4) preoperative irradiation affects such changes in the surrounding normal structures as to make the cosmetic rehabilitation with grafts doubly difficult.

We have successfully employed a V excision of the tip of the tongue with primary closure for tiny, early lesions. In aged and debilitated patients, a simple hemiglossectomy to include the primary lesion, when located at or near the edge, frequently will allow the patient to live out his remaining years in comfort. The operation may include the floor of the mouth. The wound may be left to granulate; be closed by suturing the upper and lower surfaces together, or be closed by a split-thickness skin-graft held in place by a suitable stent. V excision and hemiglossectomy are greatly simplified by the use of the electrosurgical cutting current.

Since 1948 we have combined hemiglossectomy with radical neck dissection as the procedure of choice with the above exceptions. This we believe mandatory since 25 per cent of cancers of the tongue and floor of the mouth coming in without palpable nodes will sooner or later develop neck metastases. This operation is done in one or two ways: (1) the composite operation in which the neck

dissection is begun at the clavicle and carried upward to include the mandible, floor of the mouth, half of the tongue and a portion of the pharynx when involved, en bloc; (2) an alternate method is the so-called *pull-through operation* in which the radical neck dissection is done and the mandibular attachments of the muscles of the floor of the mouth and tongue muscles are loosened. The lower skin flaps are closed and the hemiglossectomy is done by pulling the tongue down beneath the jaw through the mouth. In the pull-through operation the mouth may be closed by reconstructing the floor of the mouth with the tissues that remain or by a graft.[13] However, the composite procedure usually necessitates the use of some type of skin-grafting for better functioning of the tongue, both in speaking and eating. This can be done by the formation of a delayed double pedicle apron flap two weeks prior to the definitive operation.

At a preliminary operation, two weeks prior to the resection, a long vertical neck flap with the pedicle at the upper end overlying the mandible is raised. The lower end of the pedicle flap is turned on itself forming a full thickness apron. The top of this apron may be sutured to the soft tissues of the cheek or to the periosteum of the lingual side of the mandible, if the mandible is to be removed. A drain is passed between the flap and the turned under apron, forcing the circulation through the upper end of the apron only. The raw surface of the neck is covered with a split thickness graft. After one week, the apron portion of the graft is cut away from the main pedicle with one or two *delays*. This apron is now used to replace the floor of the mouth that will be excised with the resected specimen.[12] It also may be accomplished by lining the elevated upper (cheek) flap of the skin incision with a split-thickness skin graft. Mandibular alignment is maintained by the insertion of stainless steel bars to bridge the gap of the resected mandible, or by the use of the Haines' external fixation apparatus. Alignment also may be obtained adequately by the immediate insertion of a guide plane dental prosthesis in patients who have a sufficient number of teeth to hold clasps.

Since the inception of these procedures the authors have operated upon 53 private patients with an operative mortality rate of 11 per cent. Of these, three operative deaths can be attributed to acute respiratory obstruction. It now is the rule to do a preliminary tracheostomy prior to the operation. One death is accounted for because of nonresectability. This patient succumbed to fatal hemorrhage from the common carotid artery the wall of which was weakened by tumor invasion. We hope to increase the scope of the procedure by the free use of homo-arterial grafts in the more extensive cases. Another patient with extensive disease died of hemorrhage because the ligature on the stump of the external carotid artery cut through an atheromatous plaque on the seventh postoperative day. The sixth patient died from a coronary occlusion on the sixth postoperative day.

All patients who survived the operation and who did not develop disabling recurrences have been rehabilitated; 1 as a minister, 1 as an auctioneer, 1 as a physician, 1 as a newspaper editor and others as business men, factory workers, stenographers and housewives.

Of the total number of 53 private patients, there have been 11 recurrences; 9 of these have died. Many of these patients have been operated upon too recently to give five year survivals.

CARCINOMA OF BUCCAL MUCOSA AND LOWER GINGIVA

Carcinomas of the cheeks comprise about 9.5 per cent of all intraoral tumors, being the third most frequent site after carcinoma of the tongue and the lip. The midportion of the cheek opposite the occlusal level of the teeth is the site most frequently involved, although the disease may originate at any point. A noticeable number of carcinomas do arise near the labial commissures. Buccal lesions usually extend to involve the adjacent structures, particularly the mandible or the maxilla early in the course of the disease. Carcinomas arising in the posterior portion of the mucosa near the angles of the jaws are particularly treacherous, as they quickly involve the mandible, masseter muscles and early invade the pterygoid fossa.

Chronic irritation obviously is the most important etiologic factor in carcinoma of the cheek—more so than in any other type of intraoral cancer. The buccal mucosa is so situated as to be easily irritated by the teeth, especially by sharp or ill-fitting dentures, and tobacco. It generally is assumed that leukoplakia is a very common, precancerous lesion in the oral mucous membranes. Intraoral leukoplakia occurs most often in the mucosa of the cheek. Pre-existing leukoplakia has been reported to have occurred in anywhere from 22 per cent to 70 per cent of the cancers of the cheek.[7, 12] Leukoplakia so frequently is assumed to be such a predisposing factor in carcinoma of the buccal mucosa, that we believe very strongly that all cases of leukoplakia should be observed very carefully for long periods of time.

The most successful treatment of carcinoma of the cheek is by both irradiation and surgical excision. Small and moderate sized buccal lesions seem to give the best results by irradiation. Using an intraoral cone, 4,000 to 5,000 roentgens over a 10 day period, or 6,000 to 7,000 roentgens over a three to four week period, are the ideal dosages in our hands. Persistent or recurrent lesions are removed widely by electrosurgery. The more extensive lesions require resection of the entire cheek and adjacent involved bone, followed immediately by plastic repair, with appropriate skin flaps. Occasionally, it is necessary to resect one table of the mandible, or a portion of the maxilla. When the growth has destroyed the bulk of the soft tissues of the cheek and attached itself to the skin, the skin, of course, must be widely sacrificed. When the entire mandible is removed, the opposite mandible and chin are prevented from drifting to the operative side by the same methods employed in the composite operation.

Carcinoma occurring far back on the buccal surface near the angles of the jaws gives a persistently poor prognosis, either by irradiation therapy, combined irradiation therapy and surgery, or radical surgery.

Until recently, the authors were in agreement with the majority of their colleagues is not advising prophylactic neck dissection for carcinoma of the buccal mucosa. However, certain doubts have arisen as to the accuracy of this belief

during recent months. It usually is reported that 50 per cent to 60 per cent of buccal mucosa carcinomas do not metastasize, assuming that the primary lesion was adequately treated.[3, 7, 12] Also, it has been averred by many investigators that prophylactic radical neck dissection in dealing with this lesion should be held in abeyance until the appearance of cervical metastases. However, it should be remembered that composite and pull-through procedures are based on the assumption that 25 per cent of carcinomas of the tongue, without palpable nodes when first seen, subsequently will develop metastatic nodes. It, therefore, seems a little inconsistent to base a radical en bloc operation on a 25 per cent figure for carcinoma of the tongue and withhold the same type of prophylactic removal of lymphatic spread despite a figure of 40 per cent for carcinoma of the buccal mucosa. The authors, therefore, have begun to question the inadvisability of not doing a prophylactic neck dissection, coupled with the above mentioned treatment of the local lesion. It has been our practice to combine a radical neck dissection and en bloc removal of the local lesion when there has been involvement of the skin.

In the few complete series of cases that have been reported, the five year survival rate of patients having buccal mucosa carcinoma ranges from 9 per cent to 40 per cent.

CARCINOMA OF PALATE AND UPPER GINGIVA

Squamous cell carcinoma is the most common malignant tumor of the palate. Adenocarcinoma of the mixed tumor type is the next most frequently occurring malignancy. Sarcomas and melanomas occasionally are seen on the hard palate and upper gingiva. The authors have had several cases of cylindromas, that is, the basal-cell carcinoma of mucous membrane origin, each of which destroyed large areas of bone.

The tendency now in treating squamous cell cancer of the gingiva and hard palate is to rely upon irradiation to destroy the primary growth, and to resect the neck lymph nodes when they are palpable. However, since very heavy irradiation is necessary to eradicate carcinoma, complications of secondary irradiation necrosis of the soft parts and bone often require subsequent surgical removal of the primary site. In view of this complication, and since recurrence is frequent after irradiation, we have adopted the routine of a combined course of therapy. The primary lesion first is heavily irradiated. Then four to six weeks later it is removed electrosurgically with a wide margin.

Adenocarcinomas of the mixed tumor type should not receive preoperative irradiation since they are fairly radioresistant. Surgical removal is the only sure method of cure. Occasionally, small lesions can be excised locally with a generous margin of surrounding tissue and the wound left to granulate. If the tumor extends into the nose and maxillary sinus, a much wider removal is necessary, including as much of the maxilla as is indicated to eradicate the growth. Radium tubes are applied at the time of operation whenever there is a question as to whether the entire tumor has been removed.

Most patients having cancer of the upper gingiva come late for treatment,

and since cancer in this location invades bone early, we have made it a routine that when there is any roentgenologic evidence of invasion of the maxilla, and especially the antrum, an extraoral radical procedure will be followed. Usually at operation, the growth is found extending well up into the bone, either along the lateroantral or nasoantral wall, to a much greater extent than was demonstrable clinically. We prefer to use a modified Fergusson incision, for removal of the superior maxilla.[12] A skin incision is made with a scalpel, beginning in the middle of the upper lip, passing upward to the columella of the nose, thence around the ala on the affected side and up along the border of the nose to the level of the floor of the orbit. The incision is extended through the skin and mucous membrane of the lip down to the bone, and down to the bone for the rest of its course. The horizontal limb then extends out across the cheek at right angles to the first and at the level of the lower border of the orbit as far as is necessary for thorough exposure. The cheek is dissected off the maxilla and an incision is made in the mucous membrane of the vault of the gingivobuccal sulcus and carried back to the alveolar tuberosity. With a surgical cutting current an incision is then made through the mucosa of the midline of the palate at a safe distance from the growth, then laterally behind the tumor to the cheek. The midline of the upper jaw is cut through with hammer and chisel. Any other bony attachments are similarly severed with hammer and chisel, or bone cutters. The entire area of involved jaw is then removed. Occasionally, it is necessary to remove the entire superior maxilla in a combined gingival and antral carcinoma. If the nasoantral wall and palate are not involved, they are left untouched. If these structures are involved, the operation is extended in scope, even to exenteration of the orbit, a rare necessity. These patients are rehabilitated with a dental prosthetic appliance, which extends back to cover the wound, closes off the nasopharynx, aids in talking and prevents food from getting up into the nasopharynx or nose.

Malignant tumors of the hard palate are in difficult areas to reach with an intraoral x-ray cone and may be treated by radium held in place by a leaded resinous applicator. Small cancers of the lower gingiva, that is, those under 2 cm. in diameter, may be treated by irradiation therapy through an intraoral cone, or by radium in a leadlined applicator. However, these lesions should be removed with a wide margin electrosurgically within four to six weeks after treatment.

Cancer and malignant tumors of the soft palate are extremely rare. The majority of these are squamous cell carcinomas. All are quite sensitive to irradiation; may be treated by delivering 5,000 to 6,000 roentgens through an intraoral cone in two weeks. Occasionally, a sarcoma is encountered. These are best treated by wide surgical excision. A pedicle flap of mucosa may be created from the posterior pharyngeal wall and attached to the posterior end of the hard palate to partially close off the nasopharynx.[4] Also, a denture having an extension backward prevents fluids and food from entering the nasopharynx. Radical removal of the soft palate is a fairly simple procedure and surprisingly bloodless when the operation is done with the electrosurgical cutting current. In those

soft palatine lesions which are treated with irradiation, healing seems to take place more quickly than with operation with little deformity and surprisingly little radionecrosis.

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BOOK REVIEW

The editors of THE AMERICAN SURGEON will at all times welcome books in the field of surgery and will acknowledge their receipt in these pages. The editors do not, however, agree to review all books that have been submitted without solicitation.

Practical Fluid Therapy in Pediatrics. By FONTAINE S. HILL, M.D., Assistant Professor of Pediatrics, University of Tennessee College of Medicine, Memphis; Staff Member of the John Gaston Children's Hospital and the Le Bonheur Children's Hospital. W. B. Saunders Company, Philadelphia. 1954. $6.00.

Dr. Hill has written this book for the *busy physician* who does not have time to read, or, indeed, have access to, all the numerous publications concerning water and electrolyte balance. Written in three parts, the book attempts to bring together basic principles in the practical application of fluid therapy. Included in the book is a bibliography useful for wider reading on the subject matter presented in the text.

The first part of the book includes 95 pages on the physiology of water balance and electrolyte patterns in infants and children. The subject matter is presented in an understandable and straightforward manner with just enough detail to give the reader a comprehensive review of recent studies on electrolyte inter-relationships. Chapters included in this part of the book deal with water balance, the regulation of normal electrolyte composition in intracellular and extracellular compartments, and the role of the kidneys, lungs, skin, and gastrointestinal tract in maintaining homeostasis.

The second part of the book includes 143 pages on common clinical disturbances in which changes in water and electrolyte balance may be encountered. The therapeutic approach is adequate, although somewhat labored, and it leaves something to be desired. This is understandable to the physician who has treated babies in states of dehydration and acid-base imbalance. Generally speaking, management is not difficult for the experience; on the other hand; improper care, because certain facts are disregarded, is easily given by the beginner. Dr. Hill's discussion should be helpful in avoiding some of the pitfalls.

The third part of the book includes 20 pages on procedures commonly employed in pediatric practice in obtaining blood or serum for chemical analysis, as well as in administering blood, plasma, or other solutions by subcutaneous or intravenous routes.

H. A. WENNER, M.D

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